THE OPEN UNIVERSITY
A SCIENCE FOUNDATION COURSE

UNIT 21 GENES AND EVOLUTION

STUDY GUIDE

This Unit, the last of three on genes and evolution, has only two components: this text and the TV programme. It builds on the material of Units 19 and 20, and shows what happens to genes in wild populations of animals and plants. It is a short Unit, and you should aim to complete it in about half a week. This is to allow you to make an early start on Unit 22, which is about $1\frac{1}{2}$ Units in length.

Section 2.3 is mainly about the two examples of evolution in action described in the television programme. You need to read Section 2 up to page 9, *before watching the programme.*

By the way, in Unit 22 there is an experiment in which you will need to use some arrowroot. If you do not have any at home, you should buy some at the earliest opportunity so that you have it to hand when studying that Unit (more details in the *Introduction and Guide*). You can get it from most chemists.

1 INTRODUCTION: MENDELIAN GENETICS AND EVOLUTION

The basic principles of genetics (Unit 20) were discovered by careful breeding experiments carried out under controlled conditions. Animals and plants do not, of course, behave as tidily in the natural world as they do in the laboratory. In nature, parental generations do not conveniently divide themselves into two equal-sized groups of individuals that differ in a character for which each is pure breeding, and that helpfully mate with members of the other group to produce a uniformly heterozygous F_1 generation. Nor does one find generations in the wild that are all heterozygous at a given locus and that produce textbook three-to-one ratios among their offspring. This is not to diminish the importance of the model of inheritance that was built up in Unit 20. Indeed, without the benefit of the model, derived under the relatively simple conditions of the controlled breeding experiment, it would be impossible to begin to explore the much more complicated situation that exists in the wild.

This Unit has two main aims which build upon and bring together the material of the previous two Units. The first aim is to take the model of inheritance from the laboratory out into the real world, and to see how far it can explain the pattern of inheritance of phenotypic characters that occurs in wild populations. As you read this Unit, you will see what happens to genes generation by generation in wild populations. In doing so you will obtain a better understanding of the *genetic basis* of the theory of evolution by natural selection—and this is the second major aim of the Unit.

Section 2 examines the changes that occur at the genotypic and phenotypic levels in some fairly straightforward examples of evolution in action—beginning with the now familiar example of the peppered moth *Biston betularia* and moving on to two other illustrations developed in the TV programme. Evolution depends on the existence of genetic variation in populations, and Section 3 considers how much variation there actually is, how it is maintained, and where it comes from. Following that, in Section 4, we examine in closer detail the inevitable outcome of continuing evolution: the emergence of new species. Finally, in Section 5, the Unit concludes by taking an overview of the living world in evolutionary terms, and considers how biologists classify the enormous range of species that inhabit the planet.

GENE POOL

2 EVOLUTION IN ACTION

In Unit 19 it was established that evolution proceeds by natural selection—the central point being that certain phenotypes survive better to reproductive maturity than others. In Unit 20, we said that phenotypes are influenced by genes. How do the two aspects fit together?

2.1 PHENOTYPIC AND GENOTYPIC CHANGES DURING EVOLUTION

When studying what happens to genes in the wild during evolution, it is important to look not only at individual organisms but also at whole populations. A population, as you know from Unit 19, is defined as a group of organisms of the same kind living in the same area and capable of interbreeding. During evolution, a population changes in its phenotypic characters. This does not necessarily mean that the phenotypic character of every individual in the population is different from that of all individuals in the ancestral population—although this does happen in the course of large-scale evolutionary changes, such as the evolution of humans from their more primitive ape-like ancestors. Instead, on a much smaller scale, an evolutionary change in a population can involve a change in the *proportions* of phenotypes present in the population. For example, the rapid evolutionary change that has occurred in the population of *Biston betularia* over the last two hundred years has involved a change in the proportions of pale and dark phenotypic forms.

What does such a change in the proportions of phenotypic forms in a population involve genetically? Consider the population of peppered moths in Britain at a particular time before the start of the industrial revolution. Virtually all of the moths were pale, *typica** forms—and, because paleness is determined by the presence of two recessive genes at one locus, the genotype of those pale moths was *t t* (*t* for typical).

□ If there were *N typica* moths in the population, how many *t* genes would there be in the zygotes from which these moths developed?

■ 2*N*. Each zygote is diploid (Unit 20, Section 3.2).

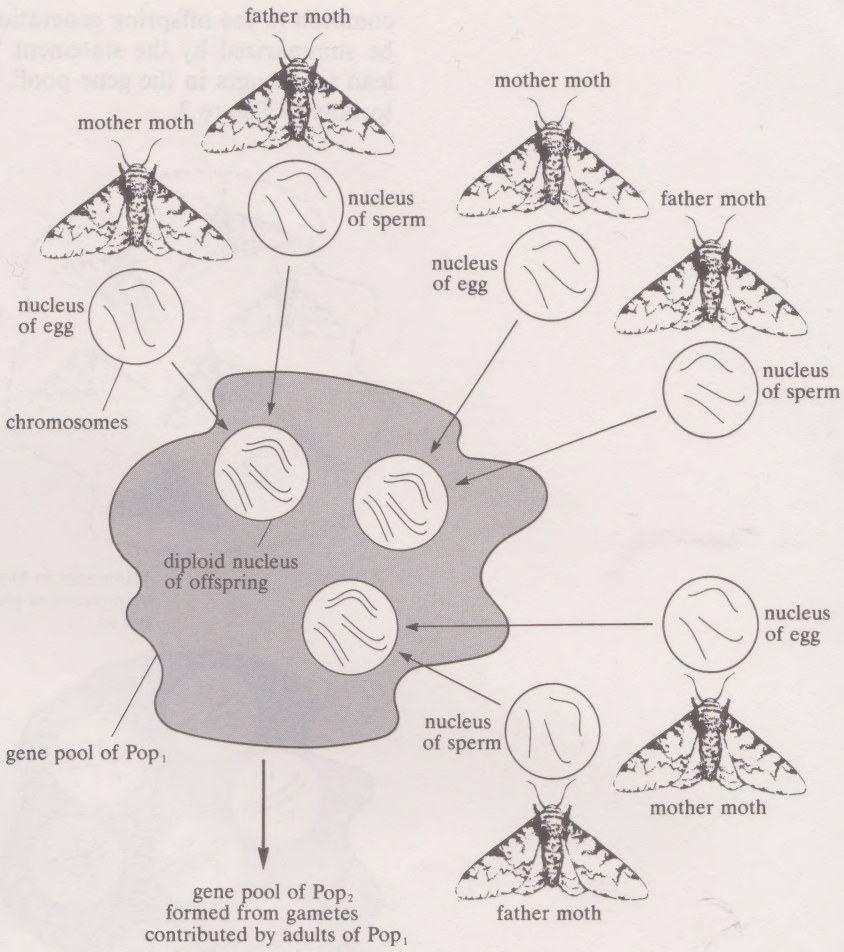
Each moth in the population (let's call it Pop_1) inherited one set of genes from its mother and one from its father, and each set includes a *t* gene. The 2*N* sets of genes that the population of *N* moths inherited from its parent population can be thought of as a reservoir of genes. The technical name for this reservoir is the **gene pool**. When the *N* moths in that population in turn produce their gametes through meiosis, the genes in these gametes are drawn from this gene pool.

These gametes, taking part in fertilization, would produce the next generation of moths—and, assuming for simplicity that all parents died—the offspring generation would constitute the new population Pop_2. The gene pool of the latter is once again formed from the inherited sets of genes. Figure 1 shows how the gene pool of Pop_1 is formed from the sets of genes contributed by the parents. The adults from Pop_1 have then contributed their genes, via their gametes, to the gene pool of Pop_2.

In the first part of the 18th century, the peppered moth gene pool (as regards genes for wing colour) would have been relatively constant, generation by generation. Almost all moths would have been pale *typica* forms; dark ones would have been terribly disadvantaged through bird predation. The gene pool would have contained an overwhelming proportion of the *t* allele. The genotype of almost all individuals would have been *t t*. Here and there, mutation would have thrown up a *T* allele (the dominant allele conferring blackness)—and a few moths would have been *T t* and even fewer would have been *T T*.

* *typica* is the technical name for the wild-type 'pale typical' form referred to in Unit 19.

FIGURE 1 The gene pool is the collection of all genotypes in a population. For simplicity, only three pairs of chromosomes are shown.



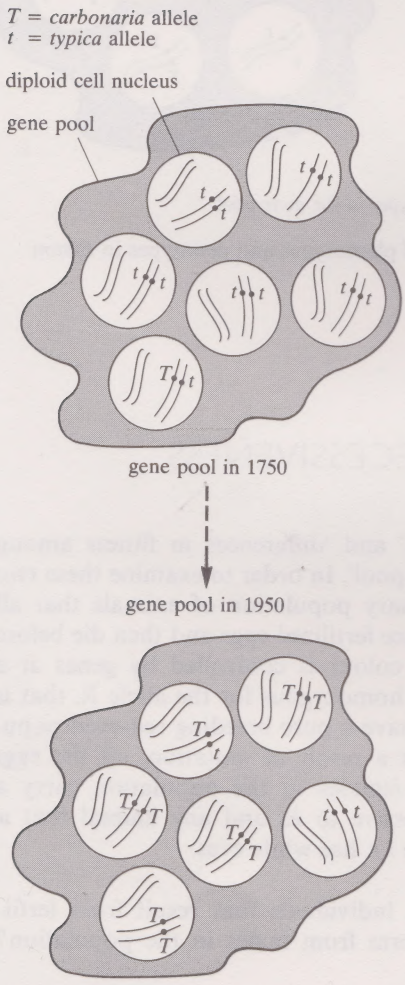


FIGURE 2 Changes in the *Biston betularia* population and its gene pool over two hundred years.

☐ What would be the phenotype(s) of *T t* moths and *T T* moths? Why would few of these have survived?

■ Because the *T* allele is dominant, moths homozygous or heterozygous for *T* are dark and are therefore *carbonaria* variants. These would have been preyed upon by birds to a great extent in the 'clean trees' era of the early 18th century.

However, as the Industrial Revolution proceeded and the environment became dirtier, the proportion of the dark-bodied *carbonaria* forms would have increased. This means that the proportion of alleles for the dark-bodied *carbonaria* moths in the population would also have increased, and the proportion of alleles for the light-bodied *typica* forms would have decreased. Thus the gene pool of the population of peppered moths in, say, 1950, will have been very different from that of the population in, say, 1750—and this is shown in Figure 2. Note how important to the subsequent course of evolution is the existence, albeit at a minimal level, of the *T* allele in the 1750 gene pool.

Evolution, therefore, has to be considered at two levels. At one level it consists of changes in phenotypes in populations over time. At another level it consists of changes of frequencies of alleles in the gene pool.

☐ From your study of Unit 20, how are these two levels linked?

■ Changes in the frequency of alleles are changes in the genotype. These produce changes in the phenotype, the fittest of which are selected.

The pale, *typica* form of the peppered moth is fitter than the dark, *carbonaria* form in a clean environment. This fact is often summarized by the statement 'natural selection acts upon phenotypes'. If one phenotype is less fit than another, fewer of its descendants will survive to reproductive maturity, and hence the allele responsible for the less fit phenotype will be less

common in the offspring generation than in the parental one. This fact can be summarized by the statement 'differences in fitness among phenotypes lead to changes in the gene pool'. These two statements are illustrated pictorially in Figure 3.

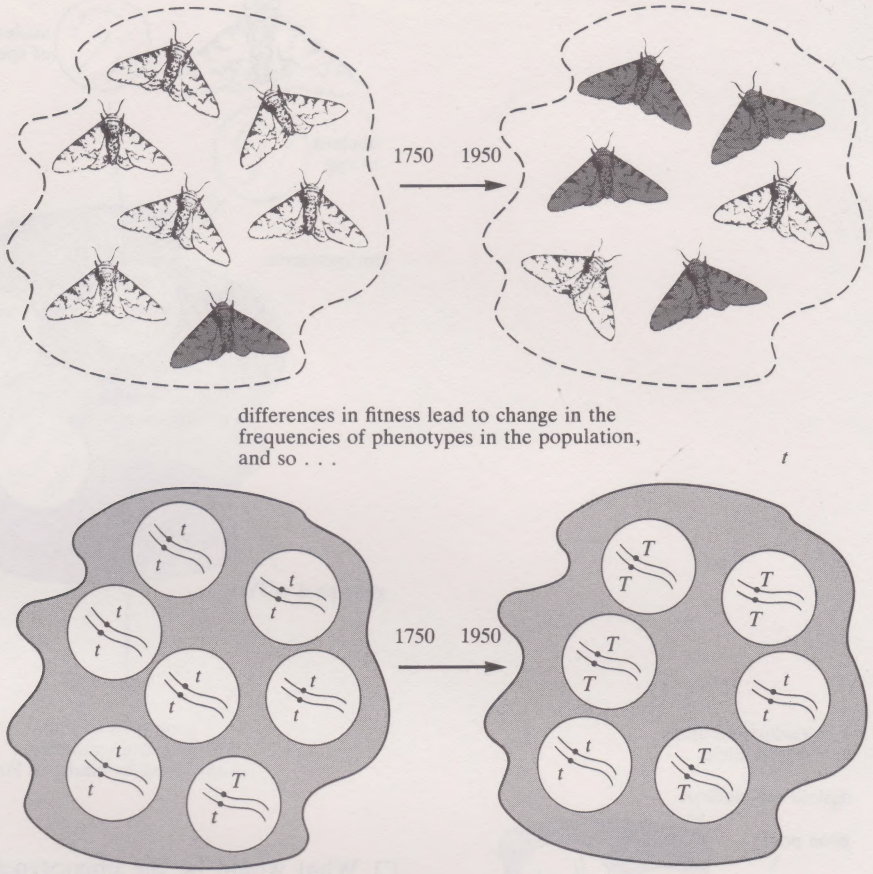


FIGURE 3 1750 to 1950: a comparison of phenotypes and genotypes in *Biston betularia.*

2.2 A CLOSER LOOK: RECESSIVENESS AND DOMINANCE

'Natural selection acts on phenotypes' and 'differences in fitness among phenotypes lead to changes in the gene pool'. In order to examine these two statements further, consider an imaginary population of animals that all have red eyes. The animals mate, produce fertilized eggs and then die before their offspring begin to develop. Eye colour is controlled by genes at a single locus, and all of the animals are homozygous for the allele *R*, that is they have the genotype *R R*. We now have a pure breeding red-eyed population. However, assume now that, as a result of mutation, all the eggs produced by one out of the million females in the population carry a mutant allele *r* instead of *R*. *r* is recessive to *R*, and any animal that is homozygous for *r*, that is with genotype *r r*, has white eyes.

☐ What will be the genotype of any individuals that result from fertilization of this female's eggs by sperm from males in the population? What colour eyes will they have?

■ The genotypes will be *R r*, because all of the males are homozygous *R R*, and so produce sperm with genotype *R*. The individuals, of course, will have red eyes because *R* is dominant and *r* is recessive.

The heterozygous *R r* individuals will therefore be indistinguishable in their appearance from the homozygous *R R* individuals. There will also be very few of them. Assuming that the eggs with the *r* allele were as successful at being fertilized and at developing into viable offspring as all of the other eggs, with the *R* allele, then about one millionth of the offspring generation should be heterozygous rather than homozygous (Figure 4).

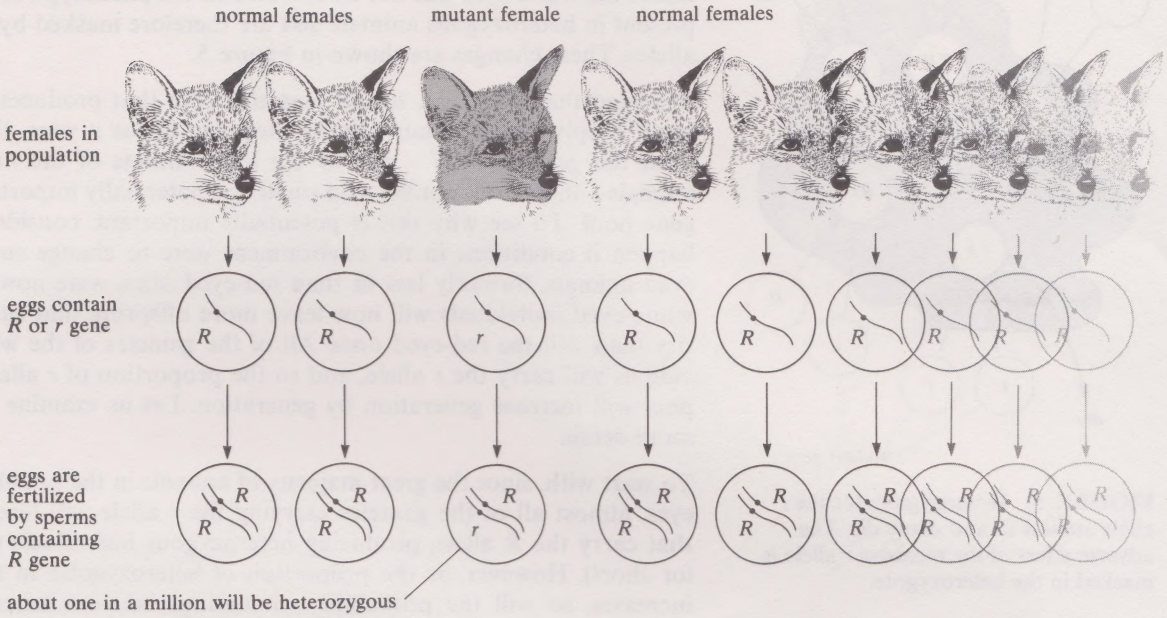


FIGURE 4 If the parental population contains one mutant female (genotype *r r*) in a million, then the offspring population (assuming all males are *R R*) will contain one heterozygote (genotype *R r*) in a million.

□ When this offspring generation in turn reproduces, is it theoretically possible for any of their progeny to have white eyes? If so, roughly what proportion of the population would they constitute? One in a million? Less than one in a million? More than one in a million?

■ Any progeny with white eyes must have the genotype *r r*, that is, they must be homozygous for the recessive allele. You know from Unit 20 (Section 4.4) that if two heterozygous individuals are crossed then approximately one-quarter of their offspring will be homozygous for the recessive character. Hence about one quarter of the offspring resulting from any matings between heterozygous individuals in this imaginary population will have white eyes. However, such matings will be exceedingly rare. Only one in a million animals in the population are heterozygous, *R r*, and the chances of *both* individuals in a mating being heterozygous is therefore much smaller than one in a million. Hence the chance of obtaining a white-eyed individual among the offspring is very much less than one in a million.

(In fact, if mating among heterozygous and homozygous red-eyed individuals occurs at random, then the chances of any one heterozygous red-eyed individual mating with another will be about one in a million multiplied by one in a million, that is one in 10^{12}.)

This example shows, therefore, that if mutations result in rare recessive alleles, the number of individuals in a population that display the recessive phenotypic character will be very low indeed. The few individuals that carry the mutant allele will almost all be heterozygous, and hence the phenotypic effect of the mutant allele will be hidden by the dominant allele. What happens, however, to the very few that are homozygous for the recessive allele and are therefore white-eyed?

Let us first consider what will happen if the very few individuals that display the recessive character are less fit than those with the dominant character—that is, in terms of the present example, if the white-eyed

FIXATION

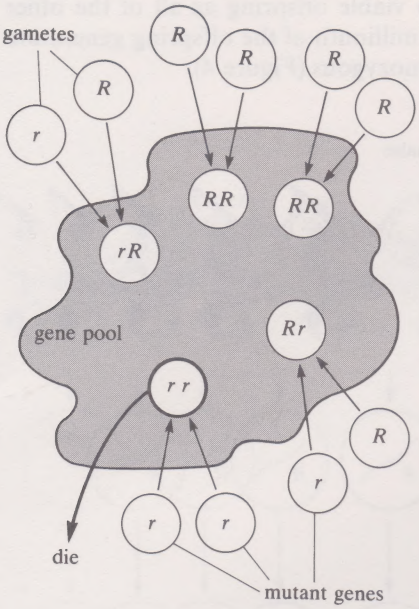


FIGURE 5 Homozygotes for the *r* allele are less fit and many die. The adverse effect of the recessive *r* allele is masked in the heterozygote.

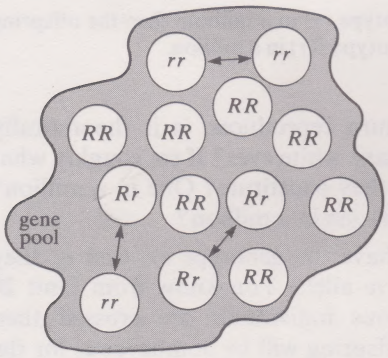


FIGURE 6 Individuals with genotypes *r r* and *R r* are now sufficiently common for frequent matings to occur between them.

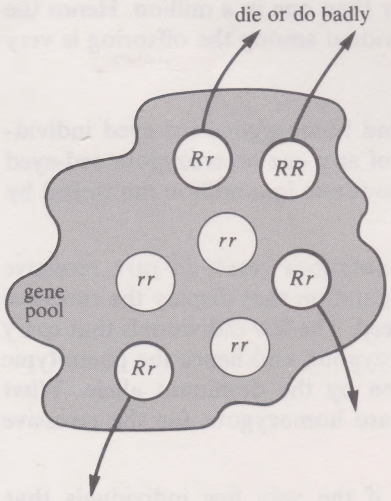


FIGURE 7 All individuals carrying the *R* allele are selected against.

animals are less fit than red-eyed ones. The definition of fitness (Unit 19) provides us with a clear answer. The white-eyed animals will leave fewer offspring that survive to maturity than will red-eyed animals, and so the proportion of alleles for white eyes in the population will become even smaller generation by generation. In fact, however, this diminution will be very small indeed, in the first place because so few of the animals have white eyes, and in the second place because the effects of the majority of the alleles for white eyes will not be revealed in the phenotype because they are present in heterozygous animals and are therefore masked by the dominant alleles. These changes are shown in Figure 5.

Provided that there is a steady mutation rate that produces a regular but small supply of the mutant allele *r*—topping up, as it were, the slight drain from the gene pool of *r* due to the lower fitness of the *r r* homozygous animals—then *r* will remain as a small but potentially important part of the gene pool. To see why this is potentially important, consider what would happen if conditions in the environment were to change such that white-eyed animals, formerly less fit than red-eyed ones, were now more fit. The white-eyed individuals will now leave more offspring that survive to maturity than will the red-eyed ones. All of the gametes of the white-eyed individuals will carry the *r* allele, and so the proportion of *r* alleles in the gene pool will increase generation by generation. Let us examine this change in more detail.

To start with, since the great majority of animals in the population have red eyes, almost all of the gametes carrying the *r* allele will fuse with gametes that carry the *R* allele, producing heterozygous individuals (heterozygotes, for short). However, as the proportion of heterozygotes in the population increases, so will the probability of matings between heterozygotes, and with them, the production of more white-eyed offspring. As generations pass, heterozygotes, and homozygous white-eyed individuals, will become sufficiently common for matings between them to become more frequent—you can see this in Figure 6. Gradually, therefore, the *r* allele will spread throughout the population, and the *R* allele will become increasingly rare. Indeed, because *R* is dominant to *r*, every individual that carries *R* is red-eyed, and so is less fit than the white-eyed animals in the population. Unlike *recessive* alleles for phenotypic characters that reduce fitness, the effects of *dominant* alleles cannot be hidden, hence any disadvantageous phenotypic effects are fully exposed and selected against (Figure 7).

Ultimately, if red-eyed individuals continue to be less fit than white-eyed ones, they will almost vanish from the population. Where once the gene pool contained a high proportion of alleles for red eyes, it will now contain an even higher proportion of genes for white ones. The *R* allele does not quite vanish from the gene pool because a few alleles for red eyes are introduced into the pool at each generation as a result of fresh mutation from *r* to *R*. However, none of the individuals carrying an *R* allele (whether *R R* or *R r*) can escape its adverse consequences and the proportion of the allele remains extremely small (Figure 8).

To summarize, a rare recessive allele for a character that confers a lower fitness on its carrier than the dominant allele can continue to constitute a small proportion of the gene pool because the phenotypic effects of the recessive allele are usually masked by the dominant allele. This gives the population considerable potential for change. This potential may well be realized if, for some reason, circumstances alter and the formerly disadvantageous recessive allele now becomes advantageous. It will spread through the population until it virtually eliminates the dominant allele, the phenotypic effects of which cannot be masked.

When one allele has spread throughout a gene pool, so that the only other alleles of it present in the gene pool arise through mutation, the allele is said to have achieved **fixation**.

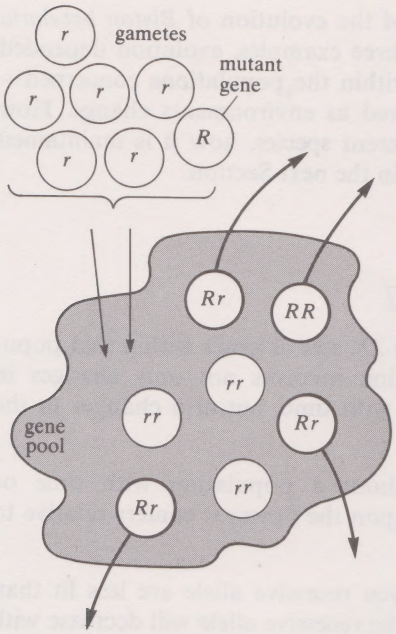


FIGURE 8 *R* alleles are not totally lost from the gene pool because some *R* is introduced into the gene pool through mutation.

2.3 CASE STUDIES OF GENETIC CHANGE IN EVOLUTION (TV PROGRAMME)

Sections 2.1 and 2.2 provide the theoretical background to the genetics of evolutionary change. This Section presents two case studies of the genetic changes that are known to accompany documented evolutionary changes in phenotypic characters. The first concerns the evolution of the ability of wild rats in Britain to withstand the rat poison Warfarin. The second concerns the ability of some plants to tolerate high levels of toxic metals in their environment. These case studies are described in the TV programme associated with this Unit. Read the points in paragraph (a) below, then watch the programme. As soon as possible after doing so, you should read paragraph (b), then go on to finish this Section.

Paragraph (a)

The aim of this programme is to show you evolution at work in two common organisms, rats and a species of grass. As you watch the programme look out for these points in particular:

(i) the use of a test to distinguish between the genetically different organisms concerned (rats and grasses);

(ii) the use of breeding crosses to study the way in which characteristics are inherited (rats and grasses);

(iii) the way in which the inheritance of the genes concerned can be explained from the results of the breeding crosses (rats and grasses);

(iv) the speed of evolution of the characteristic (rats);

(v) the way in which natural selection operates 'for' the characteristics on the mine but 'against' the same characteristic in pastures (grasses).

Paragraph (b)

In the first study, pest controllers noticed resistance to a new rat poison, Warfarin, in a rat population in Scotland. The characteristic is presumed to have started as a mutation. The allele spread through the population because natural selection was operating against rats susceptible to Warfarin—they left fewer offspring on average than resistant rats. Other populations of resistant rats developed and spread in other areas of the British Isles. These are all thought to have resulted from independent mutations.

Warfarin kills rats because it interferes with the normal mechanism of blood clotting. The blood of resistant rats clots normally, even if they have eaten the poison. This resistance is due to a dominant allele, so both the heterozygous rats and those that are homozygous for this allele are resistant. The results of breeding studies are illustrated in the programme.

The second study concerns copper tolerance in *Agrostis tenuis* (common bent grass) on an old copper mine site at Parys Mountain in Anglesey (Ynys Môn). The soil of the mine site contains enough copper to kill most of the seedling plants growing on the mine. These seedlings grow from seeds that reach the mine from surrounding areas. A few seedlings survive and grow: these are seedlings that are tolerant of the copper in the soil. Breeding tests show that copper tolerance is an inherited characteristic, although its genetics is not explained in the programme. *Agrostis* is wind-pollinated, so pollen from non-tolerant plants *away* from the mine can be carried by the wind to tolerant plants *on* the mine and thus fertilize them. However, the non-tolerant offspring from such non-tolerant × tolerant crosses are unable to grow on the mine, so a tolerant population is maintained. Pollen from tolerant plants on the mine can be blown to non-tolerant plants in surrounding pastures. The tolerant offspring of such crosses are not as fit as the non-tolerant plants and are eliminated in the highly competitive pasture environment, thus maintaining a non-tolerant population around the mine.

You will find that the two examples of evolution you have been studying, the evolution of Warfarin resistance in rats and of copper tolerance in

Agrostis tenuis, complement the study of the evolution of *Biston betularia* discussed in Unit 19. Note that, in all three examples, evolution depended on the existence of genetic variability within the populations concerned—with one or another allele being favoured as environments change. How much genetic variability there is in different species, how it is maintained and where it comes from are considered in the next Section.

SUMMARY OF SECTION 2

1 In a population of N individuals, the $2N$ sets of genes within that population is called the gene pool. Evolution involves not only changes in phenotypic make-up of the population with time, but also changes in the gene pool.

2 Whether an allele spreads throughout a population with time or becomes increasingly rare will depend upon the fitness it confers relative to other alleles.

3 If individuals homozygous for a given recessive allele are less fit than individuals carrying a dominant allele, the recessive allele will decrease with every generation. The rate of decrease is lessened by the fact that the effect of the recessive allele is masked in the heterozygote by the dominant allele. A recessive allele may therefore remain in a population at low frequencies, despite its deleterious effect when homozygous. Even so, it will eventually become very rare and settle at a level at which the rate of continuing elimination is balanced by the rate at which mutation produces new copies of it.

4 If environmental conditions change and now become such that homozygous carriers of the rare, recessive allele are now fitter than carriers of the dominant allele, the recessive allele will spread through the population, and—in the absence of mutation—would eliminate the dominant allele whose disadvantageous effects are never masked. Production of more dominant alleles by new mutation, however, maintains it at very low levels. At this point the recessive allele has achieved fixation.

5 The spread of resistance among rats to the chemical Warfarin, and the tolerance of the grass *Agrostis tenuis* to metals, provide two examples of change in the gene pool of a population resulting from natural selection.

SAQ 1 (a) Assume that the British rat population is 10^8. What is the total size of the gene pool for resistance/susceptibility to Warfarin among rats in Britain?

SAQ 2 If you are told that the allele for Warfarin resistance is dominant and that 50% of the 10^8 rats are resistant, is it possible to calculate the percentage of the allele R in the gene pool for resistance/susceptibility?

3 GENETIC VARIABILITY WITHIN POPULATIONS

This Section examines how much genetic variability there is within natural populations of living organisms. This turns out to be much higher than you might expect from a simple interpretation of the effects of natural selection, so we have to try to explain why this variability exists. You are presented with two such explanations. The Section ends by showing how recombination during meiosis increases the genotypic variability to an astonishing extent. We consider the significance of this for evolution.

In Section 2 you saw the changes that occur within the gene pool under the influence of natural selection. The examples you met showed how an allele that was formerly rare in a population can spread throughout the population if circumstances change so that the formerly disadvantageous phenotypic effect of the allele in comparison with other alleles now becomes advantageous. A formerly rare allele can sweep throughout a population until in the end it is the only allele present at its locus in the population—apart from a small input from new mutation. That is, almost every individual in the population is homozygous for that allele. As we said in Section 2.2, when this situation arises, the allele is said to have achieved fixation.

If natural selection were always to act in this way, with formerly rare alleles from time to time sweeping through the population and achieving fixation, then we would predict that over a long period of time this process would have happened to most alleles and that the great majority of loci within a population would therefore be homozygous.

The only exceptions would be the relatively few loci at which mutations had recently occurred, and the few in which a formerly rare and disadvantageous allele was now at an advantage over other alleles at the locus and was in the process of replacing them. Once an allele had achieved fixation, almost all of the individuals in the population would be homozygous for that allele and this state of affairs would persist, perhaps for millions of generations, until some other allele arose in the population through mutation and conferred a still greater advantage on those individuals that carried it.

Are populations really homozygous at most loci? Is the heterozygous condition within a population a short-lived phenomenon, spanning a few generations, as a once rare allele moves towards fixation? To answer these questions we need to look at some real populations.

3.1 GENES IN HUMAN POPULATIONS

One of the most striking things about humans is their individuality. With the exception of identical twins, no two human beings are alike. Some of this variability is due to the environment: children who have inadequate nourishment while growing up will on average have a poorer physique than children who are well fed; children who grow up in an environment where only German is spoken will end up speaking differently from children who grow up where English is the only language; and so on. Some of the variability among human beings, however, is genetic in origin. For example, there is a chemical substance phenylthiocarbamide (PTC for short) which to some people tastes very bitter, but which to others has virtually no taste at all. The difference between 'tasters' and 'non-tasters' can be considered in a normal Mendelian way: although this is something of a simplification, non-tasters are homozygous for a recessive allele, whereas tasters are either heterozygous or homozygous for the dominant allele. About 60% of the British population can taste PTC; the remaining 40% cannot.

Blood groups provide some of the best known examples of human genetic variation. If you are a blood donor, your donor card will contain two items of information about your blood group; namely, whether you are rhesus positive or rhesus negative, and to which of the blood groups O, A, B or

HETEROZYGOSITY

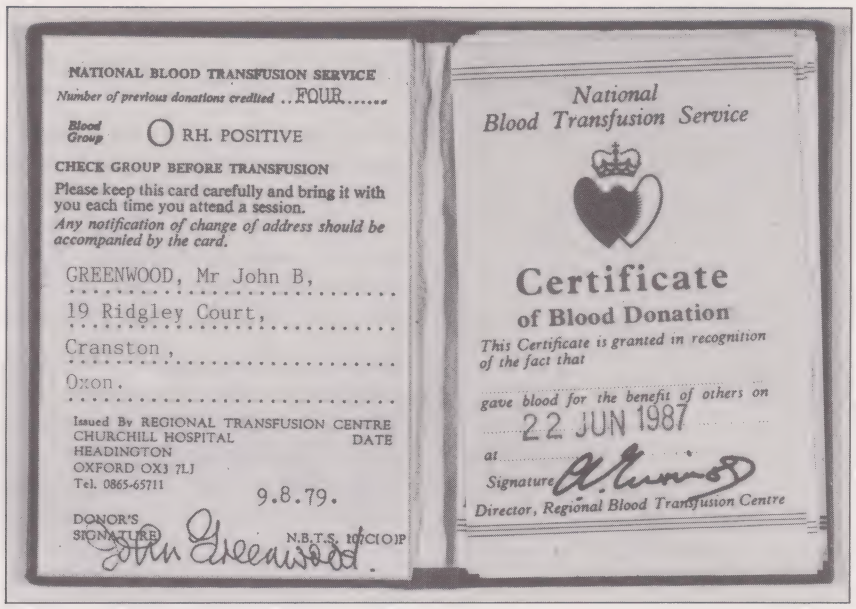

NATIONAL BLOOD TRANSFUSION SERVICE
Number of previous donations credited ..FOUR.....
Blood Group O RH. POSITIVE
CHECK GROUP BEFORE TRANSFUSION
Please keep this card carefully and bring it with you each time you attend a session.
Any notification of change of address should be accompanied by the card.
GREENWOOD, Mr John B,
19 Ridgley Court,
Cranston,
Oxon.
Issued By REGIONAL TRANSFUSION CENTRE
CHURCHILL HOSPITAL
HEADINGTON
OXFORD OX3 7LJ
Tel. 0865-65711
DATE 9.8.79.
DONOR'S SIGNATURE John Greenwood.
N.B.T.S. 10(C)(O)P
National Blood Transfusion Service
Certificate of Blood Donation
This Certificate is granted in recognition of the fact that
gave blood for the benefit of others on 22 JUN 1987
at
Signature
Director, Regional Blood Transfusion Centre

FIGURE 9 Facsimile of a blood donor card.

AB, you belong (Figure 9). Both items of information are very important. If a mother is rhesus negative and a father rhesus positive, then there is a possibility that in a second or subsequent pregnancy, the mother's immune system (the system that detects and attacks foreign objects in the body) will attack and destroy the fetus. Provided that the situation is known about, steps can be taken in advance to prevent this happening. The difference between rhesus positive and rhesus negative is relatively simple, and, at its simplest, you can think of the rhesus negative condition as arising when a person is homozygous for the recessive allele *d*, and hence has genotype *d d*. A rhesus positive person is either heterozygous *D d*, or homozygous *D D*. About 16% of white people in Europe are rhesus negative.

The ABO blood group classification is also important medically, because some of the groups are incompatible with each other, and blood transfusions between such groups are therefore not possible. Genetically, four alleles determine the ABO blood group phenotype: two different A alleles, called A1 and A2, a B allele and an O allele. The frequency of these different alleles varies from one part of the world to another. In parts of Australia, for example, more than 40% of all alleles are A1 or A2, whereas in South America, A1 and A2 are virtually absent.

These examples outline something of the genetics that underlies the variation of many features found in normal, healthy human populations. We also understand the genetics of many human diseases. Various kinds of deafness and blindness, degenerate nervous disease, muscular wasting and dwarfism all result from dominant alleles. Other diseases, such as cystic fibrosis, result from recessive alleles. Rather fewer than one baby in a hundred is born suffering from a disease with this kind of genetic origin, but everybody is heterozygous for some deleterious recessive alleles.

You can see from these examples of healthy and of diseased human conditions that genetic variation is far from rare in human populations.

☐ Is this what should be expected if natural selection acts in the way outlined at the beginning of Section 3?

■ No. If you refer back to the beginning of Section 3, you will find that we expected there to be very little genetic variation within a population. Natural selection should have eliminated all alleles at a locus except the one that confers the greatest fitness.

What of species other than our own? It might be, of course, that humans are atypical, and that all other animal and plant populations are genetically much more uniform. This explanation however, is not correct. Since 1966, when the first research in this area was published, there have been many

studies of the genetic variability of wild populations of different animals and plants, and all of them show that the populations are genetically very diverse. Look at Table 1, which illustrates this diversity.

TABLE 1 Genetic variation in natural populations of some types of animals

Organisms	Number of species studied	Average number of loci studied per species	Average proportion of heterozygous individuals per locus
(a) Invertebrates			
Drosophila	28	24	0.150
wasps	6	15	0.062
other insects	4	18	0.151
marine invertebrates	14	23	0.124
land snails	5	18	0.150
(b) Vertebrates			
fish	14	21	0.078
amphibians	11	22	0.082
reptiles	9	21	0.047
birds	4	19	0.042
mammals	30	28	0.051
(c) Average value* for these 57 invertebrate species		21.9	0.134
(d) Average value* for these 68 vertebrate species		24.1	0.060

* Average values in (c) and (d) are weighted averages. Thus, to give one example, the average number of loci studied in the 57 invertebrates is $(28 \times 24) + (6 \times 15) + (4 \times 18) + (14 \times 23) + (5 \times 18)$, all divided by 57. This, if you wish to check the full calculation, comes to 21.9. Do not worry about this detail: it is included only for completeness.

The left-hand column of the Table lists different kinds of organisms. Invertebrates are animals that lack backbones; they include insects, snails, crabs, shrimps, many different kinds of worms, and so on (see Section 5). Vertebrates are animals with backbones; they include amphibians (newts, toads, frogs and their relatives), reptiles (lizards, crocodiles, snakes, tortoises, etc.), birds and mammals.

The second column in the Table shows how many different kinds of each group of organism have been studied: 28 different kinds of *Drosophila*, 6 different kinds of wasp, and so on.

The third column of Table 1 shows the average number of loci studied in each species within the group of organisms. For example, 28 different species of *Drosophila* were studied, and on average 24 different loci were examined in each species. It might have been, for example, that in one species of *Drosophila* 18 loci were studied, whereas in a different species 29 were studied, and so on. From the Table, you can see that about 15 to 24 loci were looked at in most animals.

The right-hand column of Table 1 shows how much variability there is among the genes in a population. The measure used to indicate this variability is called the **heterozygosity** of the population. If you were to take an individual from a population in which all individuals were homozygous at every locus (that is, a completely inbred population), there would be no heterozygous loci, and the heterozygosity of the population would be nil. If, on the other hand, the population were genetically more variable, then you would find that some individuals were homozygous at a particular locus whereas others were heterozygous. The proportion of individuals in the population that are heterozygous at a particular locus is called the heterozygosity for that locus. So, if half of the peppered moths in a population were heterozygous, *T t*, for the gene controlling their colour, then the heterozygosity for that locus would be 0.5.

MUTATION RATE

SELECTIVELY NEUTRAL

GENETIC DRIFT

If you look at several different loci in a particular population, then you find that the heterozygosity varies from one locus to another. To get an idea of the overall genetic variability of the population, you simply take the average of these values. So, for example, if the proportions of individuals within a population who were heterozygous at three loci, A, B and C, were 0.100, 0.125 and 0.330 respectively, the average proportion of heterozygous individuals would be $(0.100 + 0.125 + 0.330)/3 = 0.185$. This average heterozygosity is shown in the right-hand column of Table 1.

□ If you were to examine 100 loci in the diploid cells of many animals across a range of vertebrate species, how many, on average, would you expect to be heterozygous? (Consult Table 1.)

■ Six. The average heterozygosity for all vertebrates is given as 0.060. That is, 6 out of every hundred vertebrate loci are heterozygous in the average vertebrate individual.

In fact, the average human heterozygosity is not far from this figure. It is 6.7%.

□ There are about 100 000 loci in each human diploid cell. On average, how many of these will be heterozygous?

■ 6 700. This is 6.7% of 100 000.

You can see from Table 1 that the average heterozygosity of invertebrates, at about 0.134, is about twice that of vertebrates. The value for plants (not shown in the Table) is, at 0.17, higher still. So human beings, far from being genetically *more* diverse than other organisms, as we suggested earlier, are actually *less* diverse. That is, genetic variability within populations of animals and plants is the rule rather than the exception. What do these figures mean in terms of the theory of natural selection? Remember that at the beginning of Section 3 we suggested that you would expect to find heterozygous loci only when rare alleles are produced by mutation, or when formerly rare disadvantageous alleles are now favoured and are in the process of sweeping through the population. Are the heterozygosity values low enough to be consistent with this suggestion?

To answer this question, consider first how frequently mutations occur. Look at Table 2, which shows some typical values of **mutation rates**. The term simply means the frequency with which one allele mutates to another as one generation gives rise to the next. For example, the first entry in the Table notes that the mutation rate for maize 'purple grains' is 1×10^{-5}. This means that out of 10^5 alleles for white grains, just *one* mutates to the purple grain form every generation.

TABLE 2 Mutation rates of some genes

Organism and mutation	Mutation rate
maize	
purple grains	1×10^{-5}
shrivelled grains	1×10^{-6}
Drosophila	
white eye	4×10^{-5}
mouse	
brown coat	8×10^{-6}
human	
Huntington's chorea (degenerative disease of nervous system)	1×10^{-6}
retinoblastoma (an eye disease)	1×10^{-5}

As you saw above, a human somatic cell has about 100 000 gene loci and, therefore 200 000 individual genes. The zygote, the first somatic cell, is formed from two gametes, each containing 100 000 genes derived from the

somatic cells of the mother and father. So, 200 000 genes from the parents yield 200 000 genes in each of the offspring.

□ Taking the average human mutation rate to be 1×10^{-5}, how many new mutations that were not present in its parents will a child carry in each of its diploid body cells?

■ Two. The egg from the child's mother contains about 10^5 genes. Their mutation rate is 1×10^{-5}, and so just *one* ($10^5 \times 10^{-5} = 1$) of these will have mutated and will have passed on to the child. The same argument applies to the 10^5 genes donated by the child's father. Hence, the child will possess, on average, two new mutations that its parents did not possess.

So you are likely to have two new mutant alleles possessed by neither your mother nor your father. And yet we know that the average human heterozygosity is 6.7%, which means that about 6 700 of your loci are heterozygous. Put differently, your body has somehow managed to become the receptacle for about 6 700 mutant alleles. In the introduction to Section 3 you saw that mutant alleles are usually disadvantageous to the organism in which they occur, that they are selected against, and usually disappear from the population within a very few generations. To accumulate nearly 7 000 mutant alleles at the rate of about two per generation would require these mutant alleles to remain in the population for up to 3 500 generations. This seems to rule out the idea that the genetic variability of human populations is maintained simply by the continual generation of new mutant alleles in each generation.

For the same reason, it is impossible to believe that the many heterozygous loci found in human populations represent formerly rare and disadvantageous alleles now sweeping through the population. The low mutation rate just does not allow there to be that many mutant alleles available in the population to take advantage of a change in the environment and sweep through the population in this way.

These arguments apply not only to humans but to other living organisms as well. In *Drosophila*, mice, and other organisms whose genetics are well known, the genetic variation that occurs within a population is much too high to be accounted for by the continual production, generation by generation, of disadvantageous mutant alleles. It is therefore necessary to find other explanations of genetic variability. We shall now consider two of them.

3.2 NEUTRAL MUTATIONS

One possible explanation for the genetic variability of natural populations is that when mutations occur, the mutant alleles that arise need not necessarily be either advantageous or disadvantageous to the organism. Perhaps in the conditions under which the organism is living they have no effect on its fitness at all. If that is the case, they would not be selected against, and they might persist in the population from one generation to another, instead of gradually dying out. Indeed, such mutant alleles might actually increase in the population over several generations rather than dying out.

An allele that neither increases nor decreases an organism's fitness relative to another allele at the same locus is said to be **selectively neutral**. A selectively neutral allele will fluctuate in numbers from one generation to another purely as a result of chance events—whether the organisms carrying the gene by good luck happen to reproduce well, or by bad luck happen to be eaten or struck by disease before realizing their full reproductive potential. These random fluctuations are known as **genetic drift**. Just as a log in the middle of the English Channel could end up either on the English or the French shore as a result of the random buffeting of waves and wind, so a selectively neutral allele could end up, through genetic drift, either vanishing from the population or achieving fixation.

Those scientists who emphasize the importance of selectively neutral alleles in evolution are sometimes called 'neutralists', to distinguish them from 'selectionists', who emphasize the importance of natural selection. There have been, and continue to be, heated arguments between them. Nobody disputes that alleles can be selectively neutral. The dispute is about the importance of selectively neutral alleles in evolution. Selectionists argue that they play no part in the evolution of adaptive phenotypic features, and that important adaptive evolutionary changes have resulted only from the action of selection on alleles that affect the fitness of the organisms that carry them. Neutralists take the opposite view—and the matter is an area of biological dispute.

Whatever the outcome of that particular argument, you should realize that nowadays a number of biologists would argue that we should not be looking for a *single* mechanism to explain how evolution happens, but rather for several mechanisms, of which natural selection is one. The orthodox view, however, is the one we have adopted in this Course—and this holds that natural selection is overwhelmingly the single most important mechanism behind the evolution of adaptive phenotypes.

3.3 BALANCED POLYMORPHISM

Whereas there is disagreement about the importance of neutral alleles in evolution, there is no dispute about the importance of another mechanism for maintaining a high degree of genetic variability within a population. This mechanism is called 'balanced polymorphism'—the meaning of which will become clear as we examine a medical condition in humans known as sickle-cell anaemia.

People who suffer from sickle-cell anaemia have an abnormality in their blood and are likely to die unless they receive special treatment. The average adult human body contains about a kilogram of a protein called haemoglobin, packed into millions of red blood cells. As you may remember from Unit 19, normal red blood cells are disc-shaped, and the haemoglobin that they contain is responsible for carrying oxygen from the lungs to the tissues and cells where it is needed. In people with normal blood, the haemoglobin is of a type called haemoglobin A. In people who suffer from sickle-cell anaemia, however, the red blood cells contain an abnormal kind of haemoglobin—properly termed haemoglobin S. This abnormal haemoglobin tends to crystallize when the blood becomes deoxygenated, so causing the red blood cells to split open and take on the typical 'sickle' shape that gives the disease its name. The haemoglobin from the broken cells leaks into the blood fluid and is no longer able to carry oxygen. As a result of this leakage, the patient becomes severely anaemic and usually dies very early in life. Normal and sickled red blood cells are shown in Figures 10 and 11.

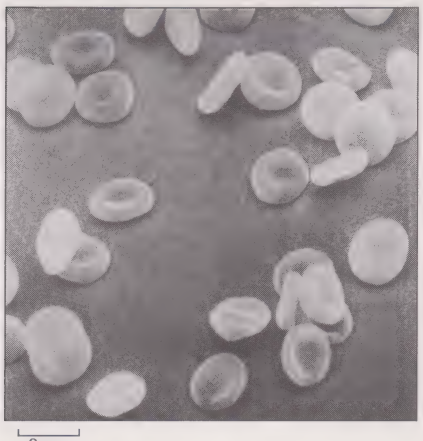


FIGURE 10 Scanning electron micrograph of normal human red blood cells.

FIGURE 11 Scanning electron micrograph of red blood cells from a person with sickle-cell anaemia.

Sufferers from sickle-cell anaemia differ at one genetic locus from people with normal haemoglobin. Whereas a person with normal haemoglobin is homozygous for the allele Hb^A at this locus and hence has genotype $Hb^A\,Hb^A$, an individual suffering from sickle-cell anaemia is homozygous at the same locus for a different allele Hb^S and so has genotype $Hb^S\,Hb^S$. (*Hb* is a standard abbreviation for the gene for haemoglobin and Hb^A and Hb^S are the conventional way of representing these alleles.)

Tens of thousands of people in the world die each year from sickle-cell anaemia, and yet the sickle-cell allele, Hb^S, is still present in the human population. Indeed, in some areas of the world, such as parts of West Africa, the Hb^S allele reaches a frequency of 10–15%. (That is, 10 to 15 out of every 100 haemoglobin genes in the population are Hb^S instead of Hb^A.) Why is the Hb^S allele so common if it has such a devastating effect on fitness? Why has it not been eliminated from the human population by natural selection? The answer to these questions becomes clear when one looks at individuals who are *heterozygous* at the locus in question: that is, who have the genotype $Hb^A\,Hb^S$. Here, the situation is rather different from most of the examples of genes we have looked at so far. Neither allele Hb^A nor allele Hb^S can be identified as dominant and the other as recessive. Instead, both are said to be *incompletely dominant*. Neither allele completely suppresses the other, and hence $Hb^A\,Hb^S$ individuals are intermediate in phenotype between $Hb^A\,Hb^A$ and $Hb^S\,Hb^S$ individuals. $Hb^A\,Hb^S$ individuals suffer from anaemia, but only mildly. A proportion, but by no means all, of their red blood cells become sickle-shaped. This, of itself, is not sufficient to explain the high incidence of the Hb^S allele in human populations. However, it has become clear that heterozygous $Hb^A\,Hb^S$ individuals do have one major advantage over individuals who are either homozygous $Hb^A\,Hb^A$ or homozygous $Hb^S\,Hb^S$ under certain conditions. They are better able to withstand malaria.

There are several lines of evidence to support this conclusion. Here we shall consider two. *First*, look at Figure 12, which shows the distribution of a particularly severe form of malaria in Africa and southern Asia. Compare this with Figure 13, which shows the distribution of the Hb^S allele in the same part of the world.

FIGURE 12 Distribution of malignant malaria caused by the parasite *Plasmodium falciparum*.

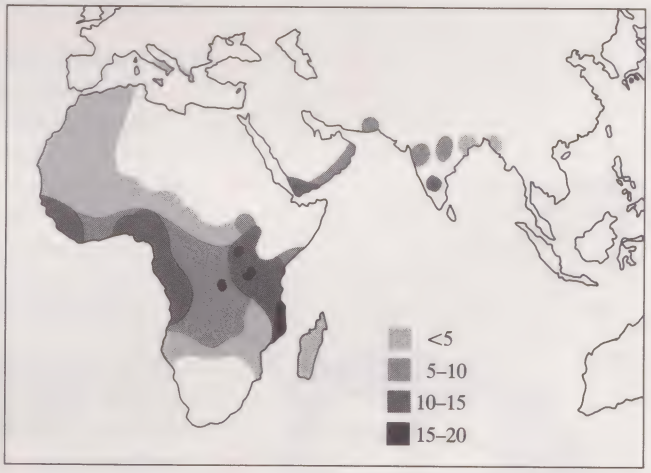


FIGURE 13 Distribution of the allele Hb^S (per cent within the human population) which, in the homozygous condition, is responsible for sickle-cell anaemia. The frequency of Hb^S is high in parts of the world where malaria is endemic because individuals heterozygous for the Hb^S allele are highly resistant to malaria.

☐ What relationship is there between the two distributions?

■ They are very similar. Areas with a high incidence of malaria, such as tropical Africa and India, also have a high incidence of the Hb^S allele. In areas where malaria is less common, such as the Sahara and southern Africa, the Hb^S allele is less common.

POLYMORPHISM

BALANCED POLYMORPHISM

Second, the parasite responsible for malaria is not able to do as well in sickle-shaped as it does in normal red blood cells. The parasite is called *Plasmodium falciparum* (a name you need not remember), and lives inside and feeds upon red blood cells (Figure 14). Since many of the sickle-shaped red blood cells have been split open and damaged by the abnormal haemoglobin, they are no longer available to the parasite, so it has fewer homes to choose from within the body. Also, even if the parasite does manage to enter an intact red blood cell, the cell may still subsequently split open. If that happens, both the damaged cell and its parasite are engulfed and destroyed by the body's white blood cells.

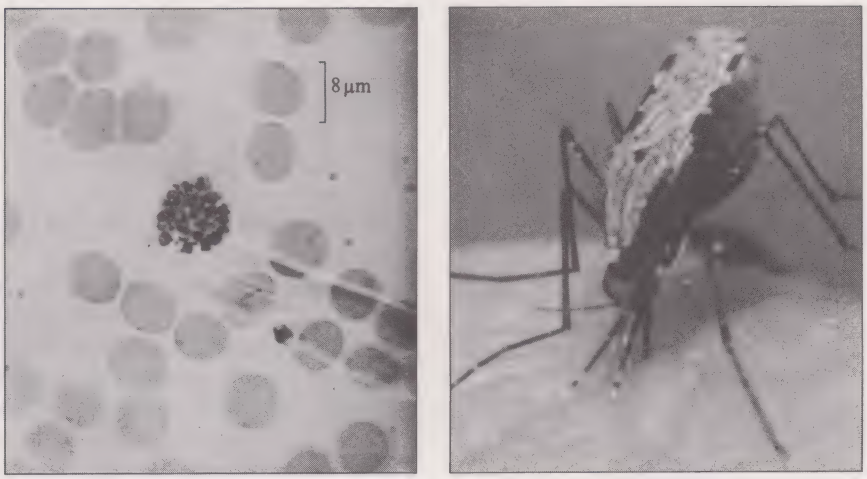


FIGURE 14 (a) *Plasmodium falciparum*, the malarial parasite. (b) *Anopheles:* the mosquito responsible for the transmission of malaria.

In malarial regions, therefore, heterozygous $Hb^A\,Hb^S$ individuals suffer from mild anaemia, but this disadvantage is more than offset by their greater resistance to malaria. This means that many of them will survive to reproductive age, will have children, and so will pass both Hb^A and Hb^S alleles on to the next generation.

☐ Consider what the genotypes of these children will be, and how this will affect their fitness. Suppose first that both the mother and father were heterozygous $Hb^A\,Hb^S$. What possible genotypes might their children have? (Think back to Unit 20).

■ There are three possible genotypes: $Hb^A\,Hb^A$, $Hb^A\,Hb^S$ and $Hb^S\,Hb^S$. Genotype $Hb^A\,Hb^A$ will occur when both parents have contributed an Hb^A allele. $Hb^A\,Hb^S$ will occur when one parent contributes an Hb^A allele and the other an Hb^S allele. $Hb^S\,Hb^S$ will occur when both parents contribute an Hb^S allele.

All (or nearly all) of the $Hb^S\,Hb^S$ individuals will die very early in life, and so their genes will be lost from the gene pool. Rather more of the children with the $Hb^A\,Hb^S$ genotype will survive to maturity and themselves have children than will children with the $Hb^A\,Hb^A$ genotype—because they are more resistant to malaria.

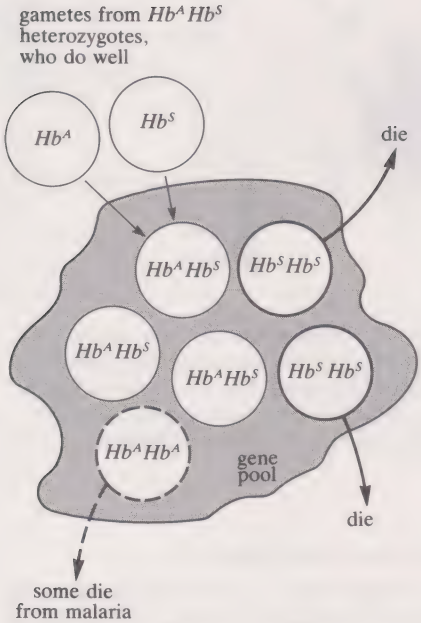


FIGURE 15 Balanced polymorphism in the Hb^S/Hb^A gene pool.

☐ Suppose now that only one of the parents is heterozygous $Hb^A\,Hb^S$, and that the other is homozygous $Hb^A\,Hb^A$. What possible genotypes will their children have?

■ They will be either $Hb^A\,Hb^A$ or $Hb^A\,Hb^S$.

The $Hb^A\,Hb^S$ children, being resistant to malaria, once again do better than those with the $Hb^A\,Hb^A$ genotype.

Finally, if both parents are homozygous $Hb^A\,Hb^A$, then all their children will be $Hb^A\,Hb^A$ as well. Look at Figure 15, which summarizes the situation. Notice that some of the Hb^S alleles are being drained off from the

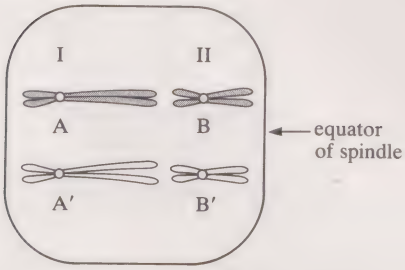
gene pool through the death of the $Hb^S\,Hb^S$ homozygotes, but that this draining effect is offset by the topping up of Hb^S alleles in the gene pool through the success of the heterozygous $Hb^A\,Hb^S$ individuals.

It is possible to show mathematically that the draining effect on the Hb^S allele due to the death of $Hb^S\,Hb^S$ homozygotes will achieve a *balance* with the topping up effect from the successful $Hb^A\,Hb^S$ heterozygotes. We ask you to take this on trust, but intuitively, it is not difficult to see why such a balance should be achieved. Imagine a population in which the Hb^S allele is very rare, living in a region (like Milton Keynes) where there is little or no malaria. Then suppose that malaria suddenly invades the area. The rare Hb^S allele will be present in the heterozygous condition, first because the chance of two rare genes being present in the same individual will be exceedingly remote (Section 2.2), and second because any $Hb^S\,Hb^S$ individuals will die before reproducing. With the advent of malaria, the heterozygous $Hb^A\,Hb^S$ individuals will do better than the $Hb^A\,Hb^A$ homozygotes, will leave more offspring, and so the Hb^S allele will become more common in the population. This trend will continue generation by generation, and so the Hb^S allele will become increasingly common. As it does so, however, the proportion of conceptions that give rise to homozygous $Hb^S\,Hb^S$ individuals will also rise, and these will die. The proportion of $Hb^S\,Hb^S$ individuals will rise increasingly rapidly as the Hb^S allele becomes more common in the population, and their death will increasingly nibble away at the advantage to the Hb^S allele derived from the resistance to malaria of the $Hb^A\,Hb^S$ heterozygotes. Eventually, there will come a point where the two exactly balance each other, and the Hb^S allele no longer increases in frequency. At this point equilibrium is reached within the gene pool.

Normal and sickle-cell haemoglobin is an example of a **polymorphism**. (The word polymorphism comes from the Greek, with the literal meaning of 'many-shaped'.) In biology, the word polymorphism is used to describe the situation where there are at least two distinctive phenotypes and genotypes in a population. In the present example, the two phenotypes are haemoglobin A and haemoglobin S. This particular kind of polymorphism, which results from a balance between a conflicting advantage and disadvantage, is called a **balanced polymorphism**.

Balanced polymorphisms of this kind are not uncommon and, to bring us back to the point from which the discussion began at the end of Section 3.1, they undoubtedly play an important part in maintaining gene variability within gene pools. As you will see from the next Section, living organisms have ways of stirring up their gene pools with very interesting consequences for the variety of genes they contain.

chromosomes I and II could line up like this at metaphase I of meiosis



or they could, for example, line themselves up like this

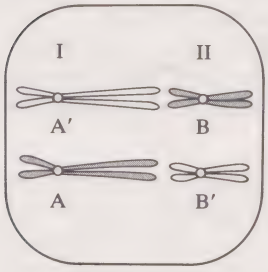


FIGURE 16 Independent assortment of chromosomes in meiosis.

3.4 STIRRING UP THE GENE POOL: RECOMBINATION

In Unit 20, Section 5.2, we introduced the term recombination. Recombination refers to the shuffling of genes that takes place during meiosis, *both* through independent assortment (which itself results from the random position that homologous pairs of chromosomes take up with respect to one another at the equator of the spindle during metaphase I of meiosis) *and* from the crossing over that occurs during prophase I of meiosis. Figures 16 and 17 revise these two processes—remember that both processes contribute to recombination.

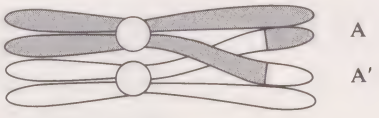


FIGURE 17 Exchange of chromosome material and hence of genes as a result of crossing over during meiosis.

Consider first the variation that arises from independent assortment. You came across a good example of this in Unit 20, Section 5.1. This described the inheritance of maize grain colour (purple or white) and texture (smooth or shrivelled), and Figure 18 summarizes how independent assortment gives rise to four genetically different types of gametes.

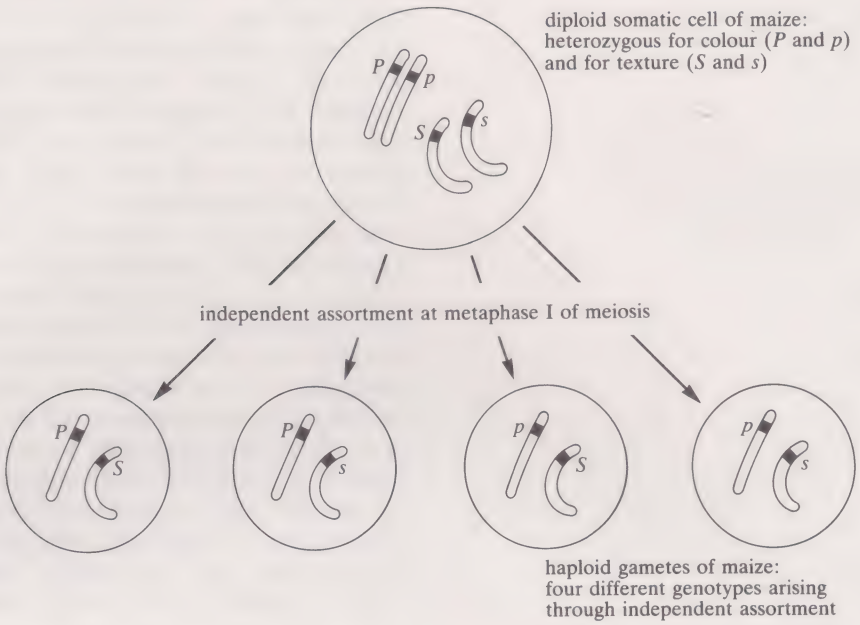


FIGURE 18 Four genetically different gametes (as regards grain colour and texture) are produced by independent assortment.

These, when cross-fertilized, produce nine genetically different types of offspring (having four distinct phenotypes in the ratio 9 : 3 : 3 : 1, as you may remember from Unit 20, Section 5.1). This is shown in Table 3.

TABLE 3 The consequences of independent assortment at a pair of loci on non-homologous chromosomes in maize which are heterozygous respectively for grain colour and texture

genotypes of both parents	*PpSs*
genotypes of gametes	*PS pS Ps ps*
genotypes of F_1 plants	*PPSS PPSs PPss* *PpSS PpSs Ppss* *ppSS ppSs ppss*
phenotypes of F_1 plants	purple, smooth grains white, smooth grains purple, shrivelled grains white, shrivelled grains

Things rapidly become more complicated when more than just two heterozygous loci are considered. If both parents are heterozygous at three loci on three different chromosome pairs, then each will produce $2^3 = 8$ genetically different types of gametes, which upon fertilization can produce $3^3 = 27$ different genotypes among their offspring. In general, if parents are heterozygous at N different loci all on different chromosome pairs, then independent assortment will produce 2^N genetically different types of gametes and 3^N different genotypes among the offspring.

We said in Section 3.1 that an average human being has about 6 700 heterozygous loci. These are carried on 23 pairs of chromosomes. Assume for simplicity's sake that the 6 700 heterozygotic loci are equally distributed among the 23 pairs of chromosomes, and that no crossing over takes place at all during meiosis. This means, in effect, that each pair of homologous chromosomes is like a single gigantic heterozygotic locus—with, therefore,

23 of these in each nucleus of a human parent. N is therefore 23 and a human parent would therefore produce $2^N = 2^{23} = 8\,388\,608$ genetically different types of gametes, which on fertilization could produce $3^{23} \approx 9.4 \times 10^{10}$ different genotypes among the offspring. That is 94 thousand million genotypically different offspring through independent assortment alone.

But this huge number is nothing compared with what we shall see when we consider the additional effect of crossing over. In the calculations we have just done for human gametes, we assumed that crossing over did not happen. This time let us assume that crossing over *does* occur—on all chromosomes and involving recombination of the genes at all of the heterozygous loci. So, if a parent had two heterozygous loci on the same pair of homologous chromosomes, it could produce four genetically different kinds of gametes. You saw an example of this in Unit 20, Section 5.2, where you read that, in *Drosophila*, the normal and cinnabar eye alleles and the normal and vestigial wing alleles lie on the same pair of chromosomes. Crossing over produces four genetically different gametes, with respective genotypes We, wE, WE and we. The mathematical argument now proceeds in exactly the same way for crossing over as it does for independent assortment: if parents have N heterozygous loci, then each will produce 2^N genetically different types of gametes, and on fertilization these have the potential to produce 3^N genetically different individuals.

If we now say that N is the average number of heterozygous loci found in a human, that is about 6700, then an individual man or woman could produce, in theory at least, 2^{6700}—that is, approximately 10^{2017}—genetically different gametes. In practice the number would be less than this because of linkage (Unit 20, Section 5.2). Nevertheless, neither you nor anyone will achieve anything like this prodigious total—which exceeds by a very long way the total number of protons, neutrons and electrons in the Universe. The number of genetically different individuals that could arise from fertilization on a cosmic scale of all these genetically different gametes would be greater still. You can see from these calculations why, with the exception of identical twins, no two human beings are ever likely to have exactly the same genotype. You can also see what an enormous reservoir of genetic variability exists in human populations for natural selection to act upon. And, in other organisms, in which the heterozygosity is higher, the reservoir of genetic variability resulting from recombination is larger still.

In talking about variation arising from independent assortment and from crossing over, it is important not to lose sight of the primary source of new genes upon which recombination depends. And that, of course, is mutation. It is helpful to think of mutation as a stream—the source of original genetic variability that feeds mutant alleles into the reservoir of the gene pool. Following this analogy, you can think of balanced polymorphism as a process that prevents these mutant alleles either from vanishing from the reservoir or from taking it over entirely. Finally, you can think of recombination as comprising the processes that stir up the gene pool, so that different individuals have different combinations of alleles, each combination conferring upon the owner its unique array of advantages and disadvantages.

Taken together, these processes of independent assortment and crossing over produce populations that have immense genetic diversity—and this diversity, accompanied by corresponding phenotypic diversity, lies at the heart of evolution.

SUMMARY OF SECTION 3

1 The theory of natural selection would at first sight lead you to predict that natural populations of organisms would be genetically rather uniform, the uniformity being only broken by a few disadvantageous mutant alleles in the process of being eliminated from the population, and even fewer newly advantageous alleles in the process of sweeping through the population.

2 Natural populations turn out to be far more variable genetically than can possibly be accounted for by these two simple explanations. The heterozygosity of natural populations is typically of the order of 5–15%.

3 Genetic drift (neutral alleles) and balanced polymorphism (selection) are mechanisms suggested to explain this genetic variability, although their relative importance is disputed.

4 Recombination, resulting from independent assortment and crossing over, vastly increases the potential for genotypic variability among individual members of a population in which some gene loci are heterozygous.

SAQ 3 Assume that the fruit-fly *Drosophila* has 10^4 gene loci and that the heterozygosity in a natural population of the fruit-fly is 0.15. If crossing over can occur between all such loci, how many genetically different kinds of gamete can potentially be produced?

SAQ 4 If malaria were to be eradicated from an area where both it and the sickle-cell allele had been common in the human population, what changes would you predict in the haemoglobin allele frequencies, and why?

4 SPECIATION

In this Section, we ask why evolution results in organisms that diverge in their features as time passes.

You now know that populations of living organisms are genetically very variable, and we have reviewed some of the reasons for this variability. There is one other major question that we need to examine, however, in our attempt to understand evolution. It is one thing to say that a population of organisms is genetically variable; it is quite another to explain why there are so many different *kinds* of organisms in the world. Lions may be genetically variable, so may tigers, leopards, pumas, jaguars and cougars; but why are there so many obviously different cat-like animals? How could they all have evolved? Why is there not just one single kind of large, cat-like predatory animal in the world? And why are there antelopes, zebras, fish, amphibia, ferns, flowering plants, seaweeds, and all of the other kinds of living organisms that were described in Unit 19? How could they have evolved into *distinct kinds* of organisms? And, bearing in mind the rather subtle differences between the *carbonaria*, *insularia* and *typica* forms of peppered moth and the rather obvious differences between tigers and domestic cats, just what do we mean by 'distinct kinds'?

To answer these questions, consider the following hypothetical example. Suppose a population of butterflies live in a tropical forest, and that they are found in every part of that forest. Suppose that the conditions in the forest have been stable for millions of years so the butterflies have become extremely well adapted to life in that environment. This means that mutant forms that arise in the population are nearly always less fit than the existing butterflies, and so do not spread throughout the population. Under these conditions no new character will evolve.

Suppose, however, that over the course of a few thousand years the climate changes and becomes very much drier, with the result that the forest dies out and is replaced by scrub or desert, and that this happens everywhere except on two or three mountains, which remain sufficiently wet for the forest to survive. (This is not fantasy; such events certainly did happen in parts of the world in the past.) What was once a well-adapted population of butterflies occupying a large, unbroken tract of forest will now be reduced to two or three small populations, confined to the forest regions on the mountain slopes. Where the forest has vanished in between, the butterflies have vanished too.

It would not be at all surprising if the conditions on two of these separate mountain tops were rather different: perhaps one mountain was further north and colder than the other; perhaps one was subject to different prevailing winds, and so on. Look at Figure 19. Suppose forest A was identical to the original unbroken forest, whereas B was colder, windier and wetter.

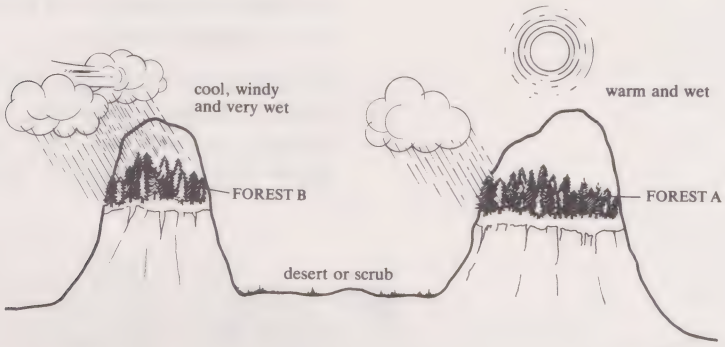


FIGURE 19 Different climates in two forest areas that were once part of a continuous tract of forest.

The population of butterflies in B might not be very well adapted to that new climate and, assuming they did not become extinct, it would not be surprising if over many generations, natural selection favoured genetically new forms of butterfly. These would then spread through the population in forest B and so the butterflies in that forest would eventually be rather different from those in A. In place of the one kind of butterfly that existed before the change in climate, there would now be two.

Suppose, now, that the climate changes yet again—with the result that the low-lying desert and scrub areas again become wetter and tree-covered, thus joining together localities A and B. The butterflies in A and B move into the newly grown forest, and meet up again. What would happen? The answer would depend upon how different the two populations of butterflies had become during their period of isolation. If they had not diverged very much, then they would probably mate with each other, and a new single, population of butterflies would be formed. Once again, there would be but one population of butterflies in the whole area. If, however, the two populations of butterflies had diverged so much that they were not able to interbreed (remember a population is defined biologically to be a group of organisms of the same kind that live in one area and are capable of interbreeding), there would now be two genetically independent populations of butterflies within the same forest. Because the two populations of butterflies could not interbreed they could not exchange genes. They would be, to use the technical term, **reproductively isolated** from one another. Once the two populations were reproductively isolated they would evolve along their own separate ways; the characters of one population would not be mixed in with those of the other. The main points of this argument are summarized in Figure 20 (overleaf).

This example shows that it is possible under one special condition for a single population of organisms to divide into two (or indeed more) genetically different populations. The special condition is that parts of the original, single population become reproductively isolated from one another. When two populations are reproductively isolated in this way they are said to belong to different **species**. The process by which new species are formed is called **speciation**. Because reproductive isolation occurs, two or more new species can arise from a population, in which formerly there was only one. And it is because of this that the world is full of so many different kinds of organisms, so many different species. If reproductive isolation did not occur, the world would contain just one species of living organism, perhaps genetically astonishingly diverse, but one species nonetheless.

In the example of the forest-living butterflies, the cause of the reproductive isolation of the two populations was a **geographical isolation** that arose

RACE

BEHAVIOURAL ISOLATION

from a change in the climate. Reproductive isolation can, however, arise for a wide variety of reasons, some climatic, some not. If, for example, disease (or fire or a predator) effectively wipes out part of the population, dividing it into two, recolonization of the intermediate area may take time. During this period the outlying population will exchange few genes with the rest. If conditions differ substantially in the areas occupied by the two groups, the effects of selection on the relative frequencies of the genes in the groups may become apparent.

Such factors encourage the formation of genetically different subsections within a single population. These are called **races**. In areas where there are already distinct races, the same factors will slow down the exchange of genes between the races. And, once there are differences between races,

1 Unbroken population of butterflies in forest.

2 Change in climate splits forest and butterfly population in two.

3 Differences in climate in the two areas result in the evolution of a different form.

4 Another change in climate causes the forest to join up again. If the butterflies are sufficiently distinct, they may not interbreed, so there are now two species. If they do interbreed there probably will be just the one species again.

FIGURE 20 A possible explanation of how populations diversify as a result of geographical isolation.

reproductive isolation may well arise, even if not immediately. When two races come together and fail to interbreed, they are by definition reproductively isolated and—again, by definition—are then considered as separate species.

Sometimes there is an intermediate stage. It may be that two races that have recently come into contact do interbreed, but the hybrids are much less fit than the pure-bred members of each race. Such hybrids will not fare well in comparison with the pure-bred organisms and, as time goes by, selection will favour those individuals who breed only with members of their own race over those who try to breed indiscriminately with members of both races. After sufficient time has elapsed, the two races may consist entirely of individuals who are completely isolated reproductively. At this point the two races have become separate species.

An example of a species that has many races is provided by the American song sparrow. In mountainous regions or offshore islands, distinct races have become isolated, each having sometimes only a few square kilometres of territory (Figure 21). In areas where geographical barriers are less pronounced, a single race may have a territory of thousands of square kilometres across which gene flow can occur relatively unimpeded.

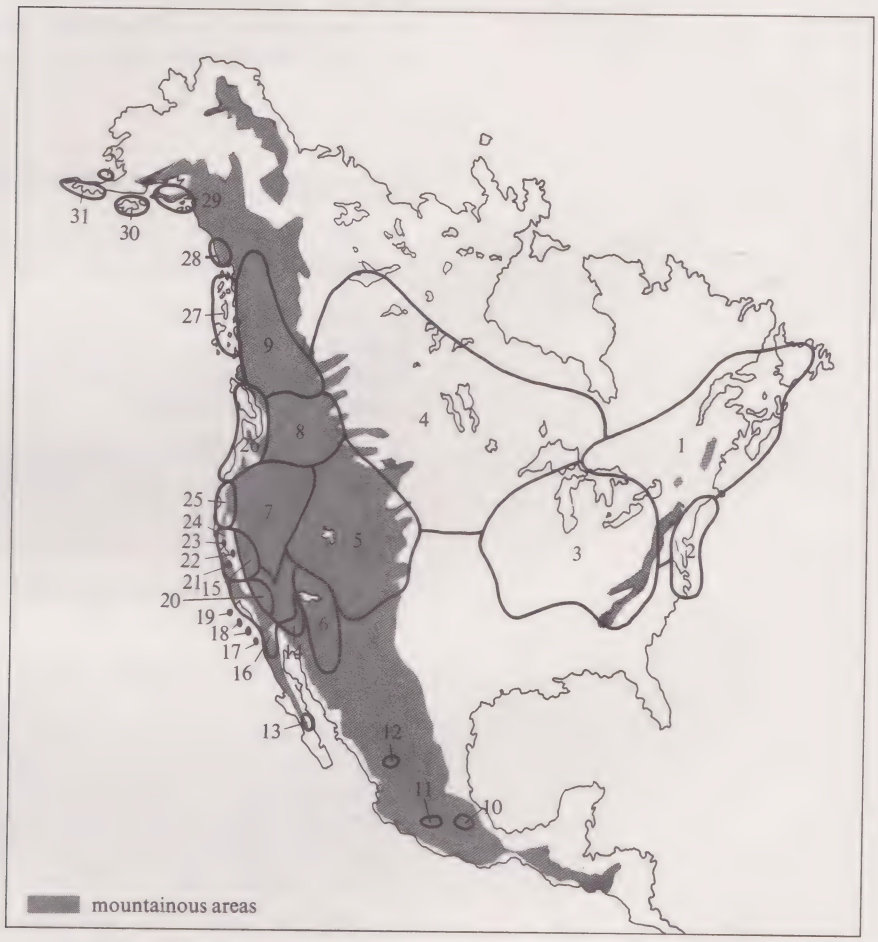


FIGURE 21 The distribution of 32 races of American song sparrow in North America.

Small differences in the markings of some kinds of bird reduce their attractiveness as mates to members of the original population. Changes in song patterns may have the same effect. If an animal changes its feeding habits, it may meet and mate less frequently with members of the main population. Races of mosquitos apparently sharing the same environment may seldom or never meet if one group develops the habit of flying and feeding by day and the other carries out these activities in the evening or at night. Reproductive isolation may thus occur as a consequence of the animal's behaviour. Such isolation is known as **behavioural isolation**. Plants living in the

TAXONOMY

TAXONOMIC HIERARCHY

same locality may be reproductively isolated if they flower at different times of the year. These, and other small differences, may effectively isolate the members of one race (or part of a gene pool) from the rest. And selection will increase the divergence until they are no longer part of the same pool and so—by our previous definition—now constitute a new species. But how can we test this in practice?

The test usually employed in deciding whether organisms that vary from one another are merely different races of the same species or whether they constitute a different species is to see if they interbreed and produce fertile offspring *under natural conditions.* Once the genetic differences have become marked, there may be no interbreeding, or else the offspring may fail to develop properly or may be infertile. Many instances of hybrid sterility are known. The mule (the offspring of a horse crossed with a donkey) is a case in point. It must be bred from a horse and a donkey each time, not from other mules. Reproductive isolation is complete; no genes will be exchanged between horses and donkeys. Sometimes, however, it is a question of degree. Domestic cattle can be crossed with American buffaloes, yaks and other relations. The hybrid bulls are sterile, but the cows are fertile when mated with domestic bulls, buffaloes or yaks. In two particular species of *Drosophila*, there is merely a lowered fertility in the hybrids; but this, coupled with other isolating factors, means that under natural conditions the two species do not exchange genes. So, even failure to interbreed is not a simple and absolute criterion to use to distinguish separate species.

SUMMARY OF SECTION 4

1 The process by which new species are formed is called speciation. Reproductive isolation is the essential feature of speciation.

2 Before reproductive isolation occurs, a population shares a common gene pool. A newly advantageous mutant allele can spread through the whole population; a formerly common allele, newly disadvantageous, can almost disappear from it.

3 The gene pool of a population can begin to separate into two (or more) pools as members of the population become geographically or behaviourally isolated (or both) from each other. Geographic or behavioural isolation always precedes reproductive isolation.

4 When there is partial separation of a gene pool, the population exists as two (or more) races.

5 Reproductive isolation means that the newly formed species go their separate ways. A newly advantageous mutant allele in one of the newly formed species will not spread to the other. An allele that comes to have a disadvantageous effect in one newly formed species may not necessarily do so in the other. As a result, it may disappear from the former but not the latter. With time, newly formed species are likely to become still more distinct from one another both in their habits and genetically, and the chance of their merging again to form one species becomes very small.

6 The formation of a new species represents a crucial step in the evolutionary process. It is no accident that Darwin's major statement on evolution was called *The Origin of Species.*

SAQ 5 If two kinds of bird—one found only in South America and one found only in Australia, but nevertheless of rather similar appearance—are caged together and found to breed, should they be classed as the same species? Give reasons for your answer.

SAQ 6 Which *one* of the statements (a)–(d) makes a correct assertion about speciation?

(a) Two races are said to have undergone speciation if they interbreed when meeting after a period of geographical isolation.

(b) Two races are said to have undergone speciation if they interbreed without experiencing a period of geographical isolation.

(c) Two races are said to have undergone speciation if they fail to interbreed when they meet after a period of geographical isolation.

(d) Two races are said to have undergone speciation if they fail to breed because they are geographically isolated and so do not meet.

SAQ 7 Which *one* of the statements (a)–(d) is the correct *explanation* of why reproductive isolation is necessary if populations of organisms are to diversify?

(a) Without reproductive isolation two races would evolve in the same direction.

(b) Reproductive isolation prevents one race from competing with the other.

(c) Reproductive isolation promotes the separate evolution of two races.

(d) Reproductive isolation creates separate gene pools upon which selection can act.

SAQ 8 Suggest three ways by which two closely related species of animals living in the same locality might be reproductively isolated by virtue of differences in their behaviour.

SAQ 9 Suggest two common practices of modern industrial society in Britain that might bring about the geographical isolation of sections of a formerly continuous population.

5 CLASSIFICATION AND EVOLUTION

The great diversity of living forms, by now a familiar idea, was introduced in Unit 19. The world that Darwin saw and was so impressed by overflows with an abundance of different species that is breathtaking. And even more impressive is evidence from fossils that, in the period from the beginning of life some 4000 million years ago, an even larger number of species, now long extinct, have lived and thrived.

In recent centuries, the work of hosts of biologists and of amateur and professional naturalists has been to categorize organisms into different species. Ideally, of course, the test they should have used was whether or not there was interbreeding under natural conditions. More often, however, they used *appearance* (the organism's morphology) as their guiding criterion. It was plain that some groups of species are quite closely related to each other and very different from other groups: cat-like creatures form a clearly related group that is very different from the group of crab-like organisms. Yet both cats and crabs, being animals, have much more in common with each other than either group has with ferns or flowering trees.

The putting together of similar species into larger groups—and then these larger groups into still larger groups—is properly called **taxonomy** (from the Greek word for 'arrangement'). And because the small groups are contained by larger groups, and these by still larger ones, the whole system of classification is termed the **taxonomic hierarchy**. As you will see shortly, the arrangement of the millions of different species into the taxonomic scheme of classification reflects *the evolutionary relationship between the members of each group*. Before looking more closely at the scheme of classification, we should explore the idea of evolutionary relationships a little further.

EVOLUTIONARY TREE

KINGDOM

SUBSPECIES

GENUS

FAMILY

ORDER

CLASS

PHYLUM

5.1 EVOLUTIONARY TREES

In Section 4, the method by which it is believed that species become distinct from one another was outlined. It needs little imagination to see that the process of divergence does not stop with the formation of species. With the passage of time, perhaps millions of years, species that are isolated from one another may become less and less similar. One would therefore expect to find among living organisms, which are, after all, the product of this evolutionary process, some species that are rather similar to each other and others that are very different from each other indeed. Refer now to Figure 22, which is an **evolutionary tree**—a semi-pictorial device that represents time vertically up the page and the evolutionary history of organisms as branches of a tree. Suppose that an ancestor of species A–F lived 100 million years (100 Ma) ago, and that shortly after that time the ancestral population split into two distinct populations (point 1). One of these populations remained relatively unchanged to the present day and forms the modern species A, but the other split and diverged several times (at points 2–5) giving rise to modern species B–F.

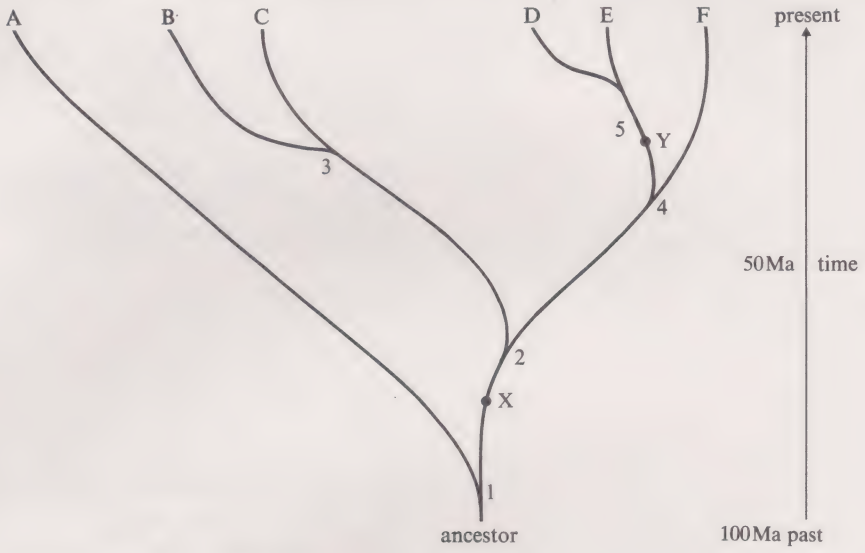


FIGURE 22 An evolutionary tree.

Species D and E diverged from each other most recently, and so are more similar to each other than either is to F. All three have more in common with each other than any of them has with B or C, and B and C have more in common with each other than either has with D, E or F. All five species B–F have more in common with each other than any of them does with A.

Note, in Figure 22, that the closeness or distance of the relationship between two species is depicted on the evolutionary tree by the length of time that has elapsed since they diverged. The horizontal separation of the lines is not usually used to depict precise extents of relatedness, although, on the whole, species that are closely related to one another (such as D, E and F in Figure 22) are usually placed closer to each other horizontally than are more distantly related species (such as C and D). The letters X and Y on the Figure are examples (taken from many) of species that existed at points in evolutionary history but that are now extinct.

Finally, note that in Figure 22 the trunk and branches of the evolutionary tree are drawn as single lines. This is the simplest way of drawing an evolutionary tree, but by no means the only way. Often broad bands are used instead of thin lines. This latter method conveys better the notion that evolution proceeds by one *population* of organisms evolving into another, rather than one individual organism evolving into another.

5.2 THE TAXONOMIC HIERARCHY

Biologists need to have a way of referring to organisms that indicates how much those organisms have in common. Therefore, as noted earlier, they have created a system that groups organisms together in a hierarchical manner. As it is impossible to be *certain* about underlying evolutionary relationships, there are several variant schemes of classification that different biologists favour; none of them is 'right' in any absolute sense. The scheme presented in this Course—based on a division of all organisms into four **kingdoms**—is one of several commonly accepted versions. The kingdoms are: animals, green plants, the fungi and prokaryotes. The approximate number of different species in each of these kingdoms is shown in the Table printed on the back of all the biology Units, and it is reproduced here as Table 4. The subdivision 'subkingdom' is a detail included for interest only.

TABLE 4 All living organisms can be divided into four kingdoms. The figures in brackets show the number of species (in thousands) in each subkingdom.

Animals	Plants	Fungi	Prokaryotes
sponges (4)	eukaryotic algae (20)	slime moulds (0.5)	bacteria (1.6)
unicells (40)	true plants (330)	true fungi (100)	blue-green bacteria (formerly termed blue-green algae) (1.5)
multicells (1 000–2 000)			

From Unit 19 you should be familiar with the idea that living organisms can be grouped into eukaryotes, organisms whose bodies contain cells with nuclei, and prokaryotes, unicellular organisms without nuclei. The many different kinds of eukaryotic organisms are separated into the animal, green plant and fungi kingdoms, whereas prokaryotes—so different from all other organisms—are assigned to their own special prokaryotic kingdom.

The question now to be explored is how this largest of all taxonomic categories, the kingdom, is subdivided. Within each kingdom are progressively smaller and smaller divisions each nestling within the other. The different-sized divisions have particular names and these are shown in Figure 23. Some intermediate categories (such as subkingdom, subclass, super-family, etc.) have been omitted from this Figure for simplicity.

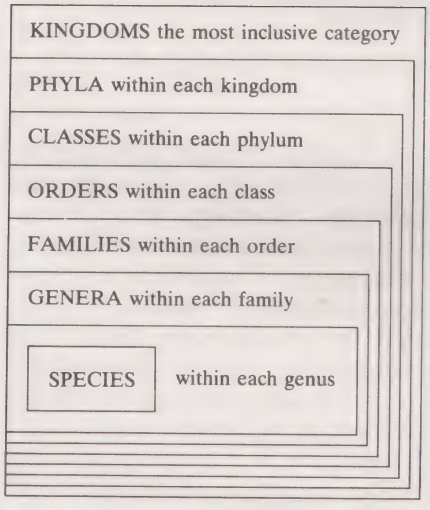


FIGURE 23 The taxonomic hierarchy.

Also missing from Figure 23 is the category **subspecies**, which is equivalent to the term 'race' that you met earlier. A subspecies differs from another in minor morphological ways—and there is also some limited restriction (often because of geographic isolation) of gene flow between the two groups. However, when and where they meet, interbreeding occurs and they therefore belong to the same species.

Groups of species that are very similar to each other in phenotype are put together in the next classificatory group, the **genus**. (The plural of this word, which is pronounced with a soft g, is *genera*.) Groups of similar genera are put together in the same **family**, and so the process goes on through **order**, **class**, and **phylum** into the largest group of all, the kingdom. The successively larger groups are created by the amalgamation of successively more diverse groups of organisms.

This system of classification was first devised by the Swedish biologist Carl von Linné (1707–1778), better known by the Latinized form of his name Carolus Linnaeus, and since his time the science of classifying organisms into taxonomic groups has developed into a substantial and intricate discipline within the field of biology. Figure 24 gives an example of how the classificatory system works in one group of animals, the arthropods; these constitute the phylum Arthropoda, one of many different phyla that form the animal kingdom.

Arthropods are animals that have the common feature of a hard, jointed skeleton on the outside of the body. (It is this skeleton that provides such an obstacle when we eat crabs and lobsters.) Do not try to remember any of the names of the arthropods in Figure 24. Simply note how the different categories fit together to form a taxonomic hierarchy. (The dashed lines indicate that the classificatory system has been simplified and that various groups have been left out.)

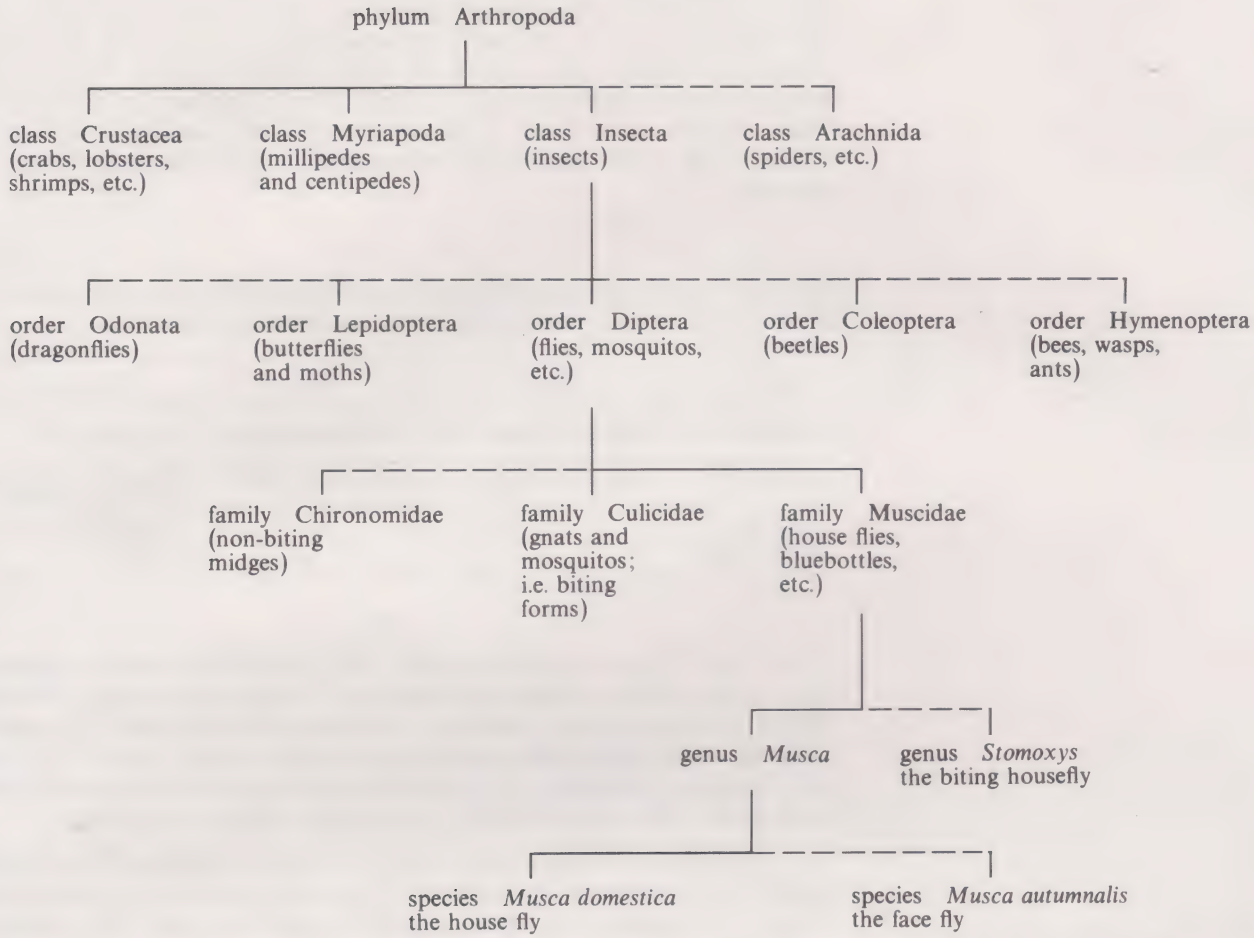


FIGURE 24 An example of the classification of a group of animals: the arthropods.

Along with this system of classification, Linnaeus devised a system for naming organisms. This system, somewhat expanded, is still used: new organisms are named along Linnaean principles, following a set of internationally agreed procedures. Each species is given two Latin names. The first of these, the generic name, indicates the genus to which the organism belongs and the second, the specific name, indicates the species. It is conventional to write the names in italics (or to underline them when handwritten), and to start the generic name with a capital letter and the specific name with a lower-case letter. When it is clear which genus is being referred to, it is often convenient to abbreviate the generic name to the initial letter followed by a full stop followed by the specific name. For example, the fruit-fly *Drosophila melanogaster* might be abbreviated to *D. melanogaster*. Sometimes, it is also useful to denote different races of a species by adding a third name after the specific name (again in italics and beginning with a lower-case letter). For example, the English race of the wren is *Troglodytes troglodytes troglodytes*, whereas the St Kilda race of the wren is *Troglodytes troglodytes hirtensis*.

□ Can you think of two main advantages of using the scientific name of an organism rather than its popular name?

■ Assuming that you are communicating with people who are familiar with the scientific name, there are two advantages in using it. First the

name is international, so one can be certain that whatever country biologists might come from they will know exactly which organism is being referred to when its scientific name is mentioned. Popular names, of course, vary from one country to another, and even within different parts of one country. Second, the scientific name tells you something about how organisms are related. For example, leopards and lions are closely related to each other, and this is immediately clear from their scientific names; they have the same generic name *Panthera*. The lion is *Panthera leo* and the leopard is *Panthera pardus*. The cheetah is not very closely related to either the leopard or the lion, and its scientific name, *Acinonyx jubatus*, reflects this, having nothing in common with the scientific names of the other two species.

Do not worry too much, incidentally, about how the scientific names should be pronounced; professional biologists themselves may use a variety of different pronunciations for the same name.

Humans belong to the genus *Homo*. There is only one living species in this genus, *Homo sapiens*, though there are several races, distinguishable from one another by differences in the frequency of certain alleles—for example, those that affect blood groups, skin colour, etc. There were, however, other species of the genus *Homo* that lived in the past but are now extinct. *Homo erectus* was one such. Members of the genus *Homo* and some other very human-like animals (all now extinct) are all grouped together in the family Hominidae. Animals belonging to this family are often called hominids.

Our closest living relatives are the apes, that is, the gorilla, chimpanzee, orang-utan, the gibbons and the siamang. They, together with other apes that are now extinct, are put in a family of their own, the Pongidae (Figure 25). Both the Hominidae and the Pongidae have many more features in common with each other than they do with other animals, and so they are grouped together in a single super-family, the Hominoidea. (Note that the ending is here -ea, not -ae.) Members of the Hominoidea are often referred to as hominoids. (Again, note the difference between hominid and hominoid.)

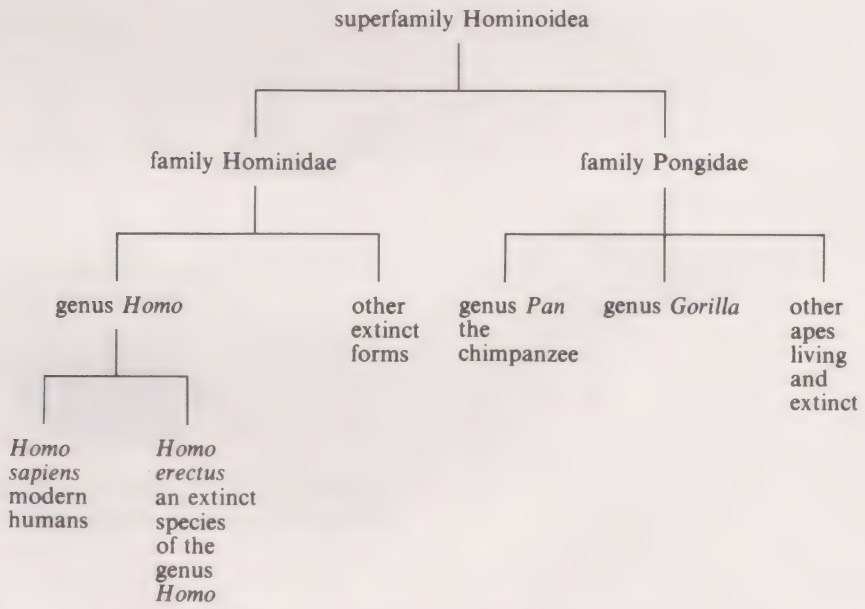


FIGURE 25 The classification of humans, apes and their relatives.

In conclusion, it is worth noting the taxonomic status of two other descriptions used in ordinary language—mammals and vertebrates. Just as the arthropods mentioned earlier were one animal kingdom phylum, another—characterized by a collection of features including a hollow nerve cord that runs along the upper side of the body—is the phylum Chordata. This in turn contains two groups, one of which is the vertebrate subphylum. And

NATURAL CLASSIFICATION

vertebrates, of course, all have a nerve cord encased in a backbone. The vertebrate subphylum contains seven (living) classes, of which one is the class Mammalia—the mammals. None of this is essential knowledge to be learned. It is included here simply so that you can get an approximate view of where we, and the other groups mentioned earlier, fit into the scheme of classification. What *is* important, and would be hard to overemphasize, is that this scheme of classification is a scheme of **natural classification**—that is one that, as far as we can be sure, describes the evolutionary relationship of the component parts. Figure 26, included only for the sake of clarity, shows a very much shortened and partial version of that scheme (for living members of the animal kingdom only).

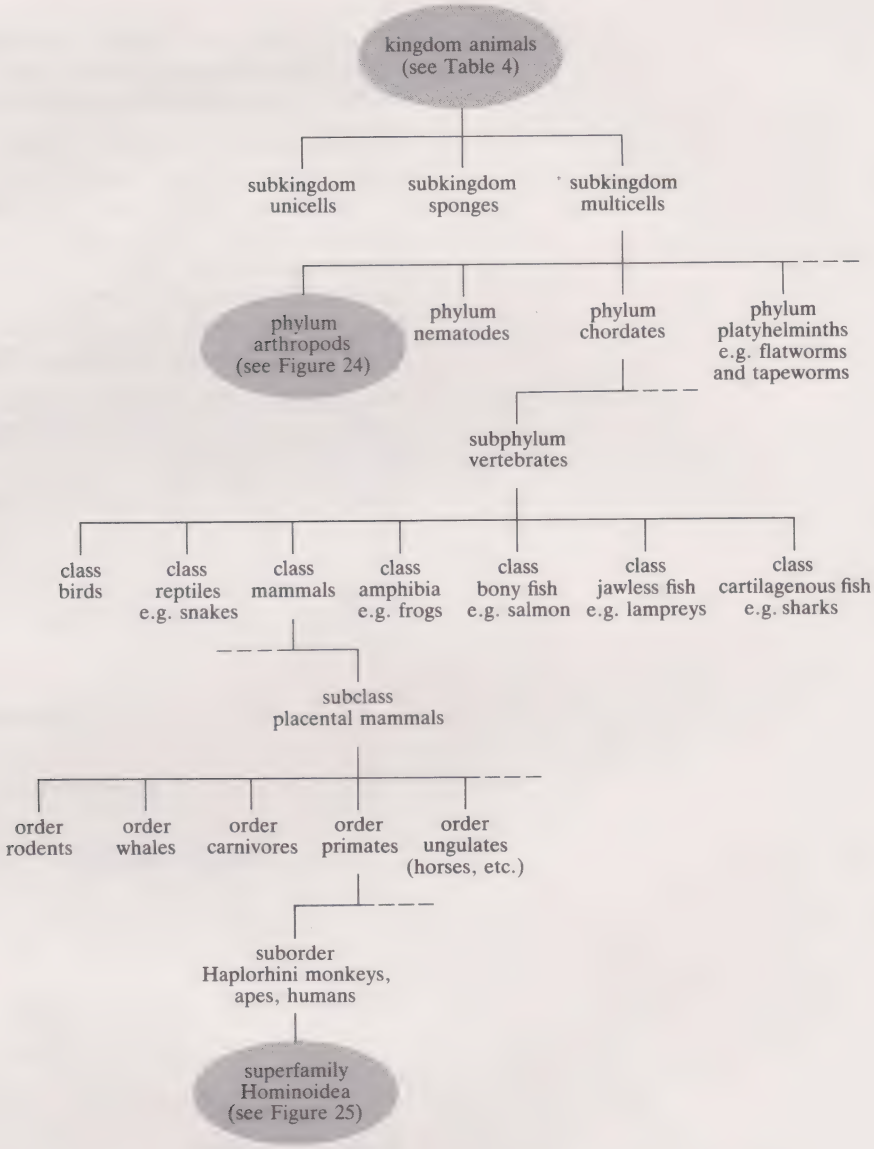


FIGURE 26 Part of the animal kingdom classification. The relationship of Table 4 and Figures 24 and 25 is shown.

5.3 WHERE NEXT?

Units 19, 20 and 21 have taken you on a journey through the world of evolutionary biology, genetics and cells. You have seen how the overall design of an organism—its phenotype—is crucially important to its ability to survive and reproduce. You have seen that genes influence phenotypic characters, and that it is inherited characters that provide the material of evolution through natural selection. You have seen also how the behaviour of chromosomes during meiosis accounts for the laws of inheritance.

But there are many questions still to be answered. If genes influence phenotypes, how do they actually do it? If the fitness of animals and plants is so important, then what goes on inside them, making them function either well, or less well? What are the processes that allow them to grow, reproduce, escape from predators, eat, digest, get energy, get rid of wastes, and all of the other things that are so vital to their fitness? These questions form the topics for the next few Units.

SUMMARY OF SECTION 5

1 The process of evolution has led to the existence today of many different kinds of organisms, some of which have only recently diverged from each other, others of which have followed evolutionarily separate pathways for hundreds of millions of years or more.

2 The relationships among different groups of organisms in evolutionary history can be depicted semi-pictorially by an evolutionary tree.

3 The evolutionary closeness of different groups of organisms is reflected in a formal system of classification, the system of natural classification.

4 Organisms are divided into progressively smaller groups that are progressively more closely related. These range from kingdoms to species including, *en route*, phyla, classes, orders, families and genera.

5 Subspecies are equivalent to races.

SAQ 10 In the evolutionary tree shown in Figure 27, A–F represent six different species. (The numbers are present for ease of reference.)

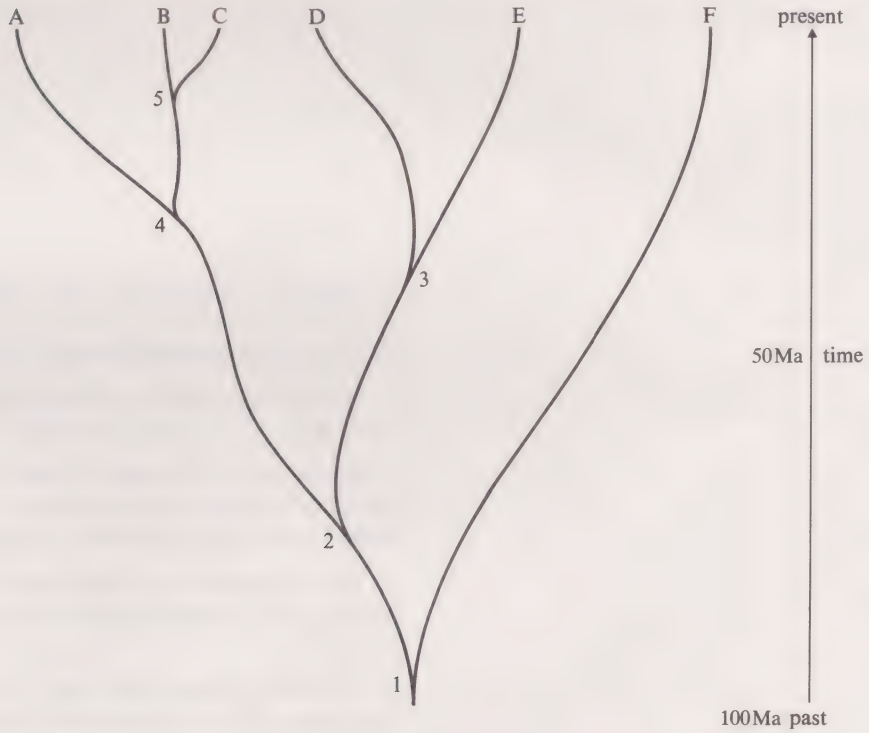


FIGURE 27 Evolutionary tree for SAQ 10.

(a) Which two of the species are the most closely related?

(b) Are D and E more closely related than A and E? Give reasons for your answer.

(c) Are C and D more closely related than B and C? Again, give reasons for your answer.

SAQ 11 (a) Is it possible to have two different genera belonging to the same family?

(b) Is it possible to have two different species within one family of plants belonging to different orders?

SAQ 12 The classification in Table 5 purports to give the complete taxonomic position of the earthworm. Change the order of the rows in Table 5 to give the correct hierarchical sequence and correct any errors in the way in which the taxonomic names have been printed. (The *content* of any given row is correct.)

TABLE 5 Incorrect presentation of earthworm classification (SAQ 12)

Hierarchical sequence	Taxonomic name	Comments
class	Oligochaeta	smooth worms, with short bristles
family	Lumbricidae	the family of earthworms and their relatives
phylum	annelida	worms with the body divided into segments
species	lumbricus Terrestris	the common earthworm
order	Terricolae	land-living worms
genus	lumbricus	earthworms

OBJECTIVES FOR UNIT 21

After you have worked through this Unit, you should be able to:

1 Explain the meaning of, and use correctly, all the terms flagged in the text (*SAQs 1, 2, 3, 5, 10, 11 and 12*)

2 Explain how a change in allele frequencies in populations depends upon the relative fitness of organisms carrying the alleles and on whether the allele is dominant or recessive. (*SAQ 4*)

3 Use information on the number of gene loci and on heterozygosity to estimate the potential genetic variability among gametes of a population. (*SAQ 3*)

4 Show how independent assortment and crossing over increase genotypic variability within a population. (*SAQ 3*)

5 Explain why reproductive isolation is necessary if populations of organisms are to diversify. (*SAQs 6, 7, 8 and 9*)

6 Demonstrate an understanding of two mechanisms (geographical isolation and behavioural isolation) by which reproductive isolation can be achieved. (*SAQs 6, 7, 8 and 9*)

7 Understand the hierarchical principle of taxonomic classification. (*SAQs 10, 11 and 12*)

SAQ ANSWERS AND COMMENTS

SAQ 1 2×10^8. Each rat has two alleles at the locus controlling susceptibility and resistance to Warfarin. Since there are 10^8 rats, the answer is therefore 2×10^8.

SAQ 2 No, you could not calculate the percentage of the *R* allele in the gene pool for resistance/susceptibility. This is because it is not possible to tell by direct observation whether a resistant rat is *RR* or *Rr*. Thus we cannot tell how many *R* alleles there are in the 50% resistant rats.

SAQ 3 We are told that the heterozygosity of *Drosophila* is 0.15. So if *Drosophila* has 10^4 gene loci, $0.15 \times 10^4 = 1\,500$ of these will be heterozygous. Provided that independent assortment and recombination occur, then 2^{1500} genetically different gametes can potentially be produced.

SAQ 4 The advantage conferred by the Hb^S gene would be lost once malaria had been eradicated. Heterozygous Hb^A Hb^S individuals would no longer be at an advantage compared with Hb^A Hb^A individuals; indeed, such individuals would be at a mild disadvantage as, having a mixture of normal and abnormal haemoglobin, they can become ill when in an oxygen-deficient environment. Homozygous Hb^S Hb^S individuals would still die early and so the Hb^S allele would gradually decline in the population (it would not be eliminated because it would be re-introduced to some extent by fresh mutation).

SAQ 5 Not necessarily. The key factor in determining whether two kinds of organism belong to the same species is whether they interbreed to produce fertile offspring *under natural conditions*. If they do, they belong to the same species.

SAQ 6 The answer is (c).

Statements (a) and (b) are wrong. If you chose either of these, re-read Section 4.

(c) is the definition of speciation given in the Section and is the correct answer.

(d) may have caused you some problems. In fact, it has caused evolutionary biologists a lot of problems too. If two races are geographically so far apart that they can never meet under natural conditions, and yet are clearly similar and perhaps even linked geographically by a series of intermediate races—surely they should be put in the same species? This is where subjective judgment and common sense come in. In this instance, the two races would almost certainly be put in the same species, just as they are for example in the case of the American song sparrow. On the other hand, if two groups of organisms live so far apart that they can never meet under natural conditions, are not linked geographically by a series of intermediate races and yet are rather similar phenotypically, it is difficult to decide whether they should be put into the same or different species. Again, it is a matter of subjective judgment. It is, in fact, comparatively rare for biologists to be able to apply the criterion of 'interbreeding under natural conditions' to determine whether the organisms they are examining belong to the same or to different species. More often than not they have to base their judgment solely on the phenotypic differences between the organisms under examination and decide for themselves how significant these differences are.

SAQ 7 (d) is the correct answer. (a) and (c) are both incorrect for the same reason: they are not explanations at all. They are simply restatements of the question in different words; that is, the question asks, 'Why is reproductive isolation necessary for diversification?' and both (a) and (c) answer 'because reproductive isolation is necessary for diversification'. This sort of pseudo-explanation is notoriously common in biology. (b) is wrong in two senses. First, it is not necessarily true that reproductive isolation prevents races from competing with one another. Second, even where it is true, it is not a correct explanation of why reproductive isolation is necessary if populations are to diversify.

SAQ 8 The two species might: (a) be active at different times of the day; (b) inhabit different parts of the same locality (different species of mosquito, for example, tend to fly at different heights within a forest); (c) avoid each other if ever they heard, saw or smelt each other; (d) might be reproductively active at different times of the year.

SAQ 9 (a) The building of roads (particularly motorways) could separate ground-living animals from each other.

(b) Agricultural practices, for example the removal of some hedgerows, could isolate the hedgerow community on one side of a field from the hedgerow community on the other.

SAQ 10 (a) B and C. Their ancestors diverged from each other more recently (at 5) than any of the other species.

(b) Yes. The ancestors of D and E diverged from each other more recently (at 3) than did those of A and E (at 2).

(c) No. The ancestors of C and D diverged at 2, whereas the ancestors of B and C diverged at 5. Note that the horizontal distance between the letters C and D is not an indicator of how closely related the two species are.

SAQ 11 (a) Yes. A family may contain several genera; see Figure 24.

(b) No. A family is a subset of an order, so all of the organisms belonging to one family necessarily belong to the same, much larger, grouping, order (just as all people living in London necessarily live in England); see Figure 24.

SAQ 12 The corrected version of Table 5 is shown in Table 6.

You should note that the generic and the specific names should be in italics (they were not in the incorrect version), and that all of the taxonomic names, except the specific name, should begin with a capital letter.

TABLE 6 Correct presentation of earthworm classification (SAQ 12)

Hierarchical sequence	Taxonomic name	Comments
phylum	Annelida	worms with the body divided into segments
class	Oligochaeta	smooth worms, with short bristles
order	Terricolae	land-living worms
family	Lumbricidae	the family of earthworms and their relatives
genus	*Lumbricus*	earthworms
species	*Lumbricus terrestris*	the common earthworm

ACKNOWLEDGEMENTS

Grateful acknowledgement is made to the following for permission to reproduce Figures in this Unit:

Figures 10 and 11 Science Photo Library, London; *Figures 14a and 14b* The Trustees of the Wellcome Trust, 1987; *Figure 21* Society for the Study of Evolution, University of Kansas.

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